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# The Factor Structure of Common Psychiatric Disorders and Their Genetic and Environmental Risk Factors in Adolescence

Jay Schulz-Heik
jay.schulzheik@gmail.com

# The Factor Structure of Common Psychiatric Disorders and Their Genetic and Environmental Risk Factors in Adolescence

by

R. JAY SCHULZ-HEIK

B.A., Colgate University, 1998

M.A., University of Colorado, Boulder, 2005

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Soo H. Rhee, Ph.D.

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Louise Silvern, Ph.D.

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Factor analyses among adults have indicated that the structure of common mental disorders may be described parsimoniously with a two factor model, with mood and anxiety disorders loading on a latent internalizing factor and antisocial behavior disorders and substance use disorders loading on a latent externalizing factor. However, little is known about the structure of mental disorders among adolescents and how posttraumatic stress and its constituent subfactors, attention deficit hyperactivity disorder, and oppositional defiant disorder would fit into such a model. Similarly, little is known about the structure of genetic and environmental influences on common mental disorders. These questions were addressed via factor analyses and multivariate twin models of a sample of adolescents aged 10 – 19 years representative of the population of Colorado (n=3867) who were assessed for eight common disorders. Factor analysis results indicated that while a two factor model fit adequately, a three factor model with attention deficit hyperactivity disorder, oppositional defiant disorder and conduct disorder forming one factor of externalizing, and substance abuse/dependence symptoms and conduct disorder forming a second factor of externalizing fit better. Posttraumatic stress disorder loaded as strongly on internalizing as the more prototypical internalizing disorders, and each of its constituent subfactors loaded more strongly on internalizing than externalizing. Twin models indicated that, in contrast to a prior study among adults,

neither genetic influences nor nonshared environmental influences could be constrained to two factors and that the best fitting model included three common genetic factors that do not conform to an internalizing–externalizing structure. These results suggest that the structure of adolescent psychopathology can be parsimoniously summarized by an internalizing–externalizing model with two factors of externalizing, and that the structure of both genetic and environmental influences do not conform to the phenotypic structure.

To My Mother

In gratitude for your support

throughout the writing of this dissertation

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Contents

The classification of psychopathology is inherently challenging, as the moods, emotions, cognitions, and behaviors that make up the dysfunctional ends of human functioning exhibits 'only modest levels of intrinsic order' (Millon, 1991). Criteria upon which a nosology may be evaluated include the extent of cross-taxa independence and within-taxa homogeneity. By these standards, the rational, categorical nosology defined in the Diagnostic and Statistical Manual (DSM; American Psychiatric Association [APA], 1994) performs poorly. Comorbidity among DSM disorders is the norm rather than the exception, and polythetic criteria sets yield extensive within-taxa heterogeneity (First, 1995; Krueger, Watson, & Barlow, 2005). Further, factor analyses of common psychiatric disorders suggest that the organization of the DSM does not accurately reflect the structure of psychopathology. Empirical evidence suggests that a dimensional, hierarchical system may perform better by these standards, and this evidence is sufficiently compelling that many researchers have called for such a system (e.g. Watson, 2005).

*The Structure of Psychopathology*

Krueger, Caspi, Moffitt, & Silva (1998) assessed the structure and stability of ten common DSM-IIIR (APA, 1987) mental disorders in a nationally representative sample of young adults in Dunedin, New Zealand. Three models of comorbidity data were tested via confirmatory factor analysis: a) a one factor solution, in which all disorders loaded onto a single factor; b) a two factor solution, in which Major Depressive Disorder (MDD), Dysthymia (DYS), Generalized Anxiety Disorder (GAD), Agoraphobia (AG), Social Phobia (SOP), Simple Phobia (SIP), and Obsessive Compulsive Disorder (OCD) loaded on an internalizing factor, and

Conduct Disorder (CD) or Antisocial Personality Disorder (ASPD; depending on the age of participant), Marijuana Dependence (MD), and Alcohol Dependence (AD) loaded on an externalizing factor; and c) a four factor solution reflecting the implicit structure of the DSM: MDD and DYS loading on a mood disorders factor, GAD, AG, SOP, SIP, and OCD loading on an anxiety disorders factor, CD/ASPD loading on a latent antisocial behavior factor, and MD and AD loading on a latent substance dependence factor. The two factor model was inspired in part by two sources. The first is the writings of Horney (1945), in which two ways of responding to the world are distinguished: approaching it (externalizing) and recoiling from it (internalizing). The second is a review of studies of the structure of child behavior (Achenbach, 1978) which found extensive support for broad-band overcontrolled and undercontrolled factors. The two factor model fit best at both ages 18 and 21. Further, the structure was stable over time, with participants' internalizing scores correlating .69 over time and externalizing scores correlating .86 over time.

Researchers also have examined the fit of a three factor model, in which the internalizing factor is divided into an anxious-misery factor (i.e. MDD, DYS, GAD) and a fear factor (e.g. PD, AGP, SOP, SIP, etc.). Krueger (1999) tested several models in the National Comorbidity Survey (NCS), a large (n=8098) nationally representative sample of the United States population. The three factor model fit best among the full sample, both random halves of the sample, men, and women, regardless of whether lifetime or prior year diagnoses were used. The lone exception was a treatment seeking subsample (n=251), in which a two-factor solution that did not split the internalizing factor into anxious-misery and fear factors fit better than the

three factor solution. The three-factor model also fit best in a large (n=7076) sample representative of the Netherlands (Vollebergh et al., 2001). The model exhibited high differential stability, as individuals' scores on the latent dimensions correlated between .85 (anxious-misery) and .96 (externalizing) over a one year interval.

A two factor model consisting of internalizing and externalizing factors best fit data among a sample of 1283 middle aged adults that was representative of the population of Minnesota (Krueger, McGue, & Iacono, 2001). A model in which the internalizing factor was split into two subfactors was not tested. This study contributed to the literature in two novel ways. In contrast to most prior studies which relied on dichotomous classification of disorders, which prevents testing the assumption that a continuous liability underlies the observed distribution of disorders, ordinal variables were examined and the multivariate contingency table did not deviate from a bivariate normal distribution.

The cross-cultural nature of the latent structure of psychopathology was more rigorously examined in the World Health Organization Collaborative Study of Psychological Problems in General Health Care (Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003). Adults in fourteen countries (Ns ranged from 196 to 800) across five continents were assessed for seven syndromes via the Composite International Diagnostic Interview (CIDI; World Health Organization, 1989). Exploratory Factor Analyses (EFAs) in most countries yielded internalizing and externalizing factors, though certain countries yielded internalizing, externalizing, and somatization factors. When fitting confirmatory factor analysis (CFA) models to all countries simultaneously, the best fitting model consisted of

internalizing and externalizing factors, and factor loadings (but not intercepts) could be constrained across countries.

There has been relatively little prior research on gender differences in the structure of psychopathology. Of the five studies reviewed above, only one (Krueger, 1999) addressed potential gender differences. In this study, it was shown that a three factor model which splits internalizing into anxious-misery and fear subfactors fit better than one-, two-, or four-factor models, both in the full sample and in separate male and female samples. It was not assessed whether parameter estimates could be constrained to be equal without a statistically significant decrease in fit, but it was noted than the difference between male and female parameter estimates ranged from .00 to .10.

Prominent features of these studies are that: 1) common DSM-defined disorders have a two factor structure, with mood and anxiety disorders falling on an internalizing factor and substance use and antisocial behavior-related diagnoses falling on an internalizing factor; 2) when tested, models that distinguish anxious-misery and fear subfactors of the internalizing factor are supported; 3) this structure is robust across genders, young adult and older adult populations, multiple longitudinal assessments, lifetime and prior year diagnoses, a wide variety of western and non-western cultures, and the DSM and ICD diagnostic systems; 4) there is little support for the implicit structure of the DSM, suggesting that cross-category comorbidity could be reduced by reorganizing the DSM; 5) disorders varied in the amount of variation that is explained by the higher order factors; 6) all but the most common

disorders were excluded; and 7) all analyses were conducted on the disorder level rather than the symptom or symptom cluster level.

Krueger et al. (1998) and Krueger (1999) argued that these models 1) make sense of comorbidity, 2) make sense of the positive association between severity and comorbidity (if observed disorders are indicators of placement on a latent factor, then more severe individuals are likely to evince more comorbidity), 3) are consistent with similar etiologies for different DSM disorders, such as shared genes, 4) predicts effectiveness of the same treatments across DSM disorders, such as selective serotonin reuptake inhibitor pharmacotherapy for internalizing disorders, and 5) suggest that the study of common disorders should focus on their common substrates: broad, higher order latent internalizing and externalizing dimensions.

Based in part on studies of the structure of psychopathology such as these, several dimensional models of psychopathology have been proposed. The authors of the DSM-IV (APA, 1994) acknowledged that a prospective dimensional model may be preferable to its present categorical model, noting that a dimensional model could increase reliability and communicate more information by capturing subthreshold attributes. However, they noted that there is no consensus on the optimal dimensions to be used.

Clark (2005) suggested that personality should serve as the basis for a nosology and argued that positive emotionality, negative emotionality, and disinhibition are the crucial facets underlying psychopathology. Krueger, Markon, Patrick, and Iacono (2005) argued that substance use and antisocial behavior disorders reflect a single underlying normally distributed continuum of risk for

externalizing disorders should be grouped together under the rubric of ‘externalizing disorders.’ Similarly, Watson (2005) argued that mood and anxiety disorders should be grouped together into ‘emotional disorders’, with three subfactors representing bipolar disorders, distress disorders, and fear disorders. Zinbarg and Barlow (1996) argued for a model with higher-order anxiety trait vulnerability and six different lower order factors.

Clark and Watson (1991) proposed that depression and anxiety be measured via a ‘tripartite’ hierarchical structure in which measurement of general distress, which is common to both depression and anxiety, is supplemented by measures of physiologic hyperarousal, which is specific to anxiety, and anhedonia, which is specific to depression. Mineka, Watson, and Clark (1998) identified weaknesses in the tripartite model, arguing that physiological arousal is indicative of panic disorder only, disorders vary in the extent to which a higher order factor explains disorder-level variation, and negative emotionality is ubiquitous across psychopathology. They suggested that more complex, multilevel hierarchical models, where many symptoms reflect multiple disorders, may be required. Much further research would be required before a complete hierarchical, dimensional nosology could be specified.

*The Placement of PTSD in a Nosology*

PTSD consists of a relatively diverse array of symptoms that are defined by a common reaction to a traumatic etiological event rather than their phenomenological similarity. It has been shown to be particularly heterogeneous (Simms, Watson, & Doebbelling, 2002; Miller, Kaloupek, Dillon, & Keane, 2004) and comorbid with many disorders (Kessler et al. 1995; Blanchard, Hickling, Taylor, Loos, & Gerardi,

1994). Many individuals who have experienced trauma evince at least one PTSD symptom, significant distress, impairment, and help-seeking despite not meeting full criteria for the disorder.

The DSM-IV task force on PTSD considered placing PTSD in three sections: anxiety disorders, dissociative disorders, and a prospective stress-response disorders class that would include Acute Stress Disorder and Disorders of Extreme Stress Not Otherwise Specified (Davidson et al., 1994). The committee evaluated these options according to the extent of perceived phenomenological similarity between PTSD and other disorders, rates of comorbidity with other disorders, similarity in etiology with other disorders, and with which disorders PTSD clinicians will most frequently need to make differential diagnoses. Their review argued that: 1) PTSD rates of comorbidity were not higher with anxiety disorders than with other classes, 2) PTSD shares phenomenological features with each class of disorders, 3) hyperarousal, intrusions, and dissociation can be found in anxiety disorders, 4) intrusions and numbing may be conceptualized as either an anxious and avoidant or dissociative processes, 5) avoidance is found across anxiety disorders, 6) effective treatments for PTSD have been derived from treatments for anxiety disorders, 7) amnesia and flashbacks are prototypical dissociative processes, 8) dissociative disorders may be due to traumatic experiences, 9) PTSD shares etiology with stress response disorders, and 10) little work has been done relating and distinguishing stress-response disorders. On the basis of these considerations, they recommended that PTSD be classified in a new “disorders related to psychological trauma” category. They also recommended that PTSD should remain in the anxiety disorders category if their

primary recommendation is not followed. Their primary recommendation was not followed, and PTSD remained in the anxiety disorders category.

Although a majority of adults have experienced a traumatic event and 8% of men and 20% of women who have experienced a traumatic event met criteria for PTSD at some point (Kessler et al., 1995), relatively little is known about how posttraumatic distress could be defined in a hierarchical, dimensional nosology. Questions that need to be addressed include: Where would it be placed in a two- or three-factor model of psychopathology? How strongly does it load on these factors? Which symptom clusters are relatively specific to individuals who experienced a traumatic event? Which symptom clusters are more shared with other disorders? What is the etiology of these relationships?

Several studies examining the structure of psychopathology have included PTSD. Cox, Clara and Enns (2002) conducted a follow up study of the 5,877 participants who completed Part II of the NCS. The ten disorders studied by Krueger et al. (1999) and PTSD were subjected to an EFA. The three factor solution was replicated, with PTSD loading more strongly on the anxious-misery factor (rotated factor loading -.39) than the fear (.10) or externalizing (.14) factors, though the other anxious-misery disorders (i.e., DYS, GAD, and MDD) loaded much more strongly on that factor (range -.64 to -.83).

A large (N=10,641) population-based study of the structure of psychopathology that included PTSD was conducted in Australia (Slade & Watson, 2006). Following a preliminary EFA and Cox et al. (2002), PTSD was included in the anxious-misery factor. The three-factor model, with anxious-misery and fear

factors conceptualized as subfactors of an internalizing factor, provided the best fit to the data for both DSM and ICD diagnoses. DSM-IV PTSD (.83) loaded equally as strongly as the other anxious-misery disorders (range .75 - .85). Interestingly, ICD-10 PTSD loaded less strongly (.69). The DSM-IV PTSD emotional numbing symptoms are absent from the ICD-10, suggesting that these symptoms may be driving much of the association between DSM-IV PTSD and other anxious-misery disorders.

In contrast to these factor analyses, latent class analysis (LCA) of common disorders was tested using the US National Comorbidity Survey Replication, a large (n=9282) nationally representative survey (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). An EFA yielded two factors labeled internalizing and externalizing. Interestingly, Oppositional Defiant Disorder (ODD) and ADHD loaded much more highly on the internalizing factor than the externalizing factor. Intermittent Explosive Disorder loaded weakly on both factors. PTSD was included in the study and loaded .64 on the internalizing factor and .16 on the externalizing factor. These loading were similar to those for some disorders that are thought to typify internalizing disorders, such as GAD and SOP. Importantly, the multivariate distribution of disorders differed significantly from the structure suggested by factor analysis results. Therefore, LCA was employed rather than CFA. Seven classes were found; generally, they reflect a progression from a large class with no disorders to increasingly rare, severe, and comorbid classes. However, there were notable inversions. For example, PD and phobias were less comorbid with externalizing disorders, perhaps reflecting a protective effect through risk aversion.

*The Structure of PTSD*

Whether individuals with posttraumatic stress disorder (PTSD) differ qualitatively or only quantitatively from more normative stress reactions has been debated. A series of taxometric analyses converged on the conclusion that PTSD represents the upper end of a continuum of stress responses rather than a discrete clinical syndrome (Ruscio, Ruscio, & Keane, 2002). Given the diverse array of symptoms of PTSD and its heterogeneity (Simms et al., 2002; Blanchard et al., 1994; Kessler et al. 1995; Miller et al., 2004), many factor analytic studies of PTSD symptoms have been conducted. The DSM-IIIR (APA, 1987) and DSM-IV (APA, 1994) define three clusters of symptoms of PTSD: reexperiencing (which is often referred to as intrusions), avoidance and numbing, and hyperarousal. Although avoidance and numbing may have the similar goal of regulating exposure to intrusions, empirical research, in addition to the theoretical arguments advanced by Foa, Zinbarg, and Rothbaum (1992), suggest that avoidance and numbing are distinct processes (for a review, see Asmundson, Stapleton, & Taylor, 2004). Only numbing symptoms predict treatment outcome (Taylor et al., 2001), different treatments show different effectiveness for avoidance but not numbing (Taylor et al., 2003), numbing is more highly correlated with depression (e.g. Asmundson, Stein, & McCreary, 2002; Taylor, Kuch, Koch, Crockett, & Passey, 1998), and only numbing is correlated with a P300 amplitude, a measure of attentional allocation (Felmingham, Bryant, Kendall, & Gordon, 2002). Therefore, a four factor model of PTSD consisting of reexperiencing, avoidance, numbing, and hyperarousal factors has frequently been compared to alternative models via CFA and found to be the best fitting model (e.g.

Taylor et al., 1998; Buckley, Blanchard, & Hickling, 1998; Asmundson et. al, 2000; King et al., 1998; Palmieri & Fitzgerald, 2005).

Two EFAs of PTSD symptoms converged on the interesting finding that the two most prototypical hyperarousal symptoms (D4: hypervigilance, D5: exaggerated startle) did not segregate with the remaining hyperarousal symptoms but instead segregated with an intrusions/avoidance factor (Simms & Watson, 1999; Taylor, 1998). Simms et al. (2002) argued that the remaining hyperarousal criteria (D1: sleep disturbance, D2: irritability, D3: difficulty concentrating) are less indicative of hyperarousal and more indicative of general distress. Accordingly, they tested a model in which the three less prototypical hyperarousal criteria (D1: sleep disturbance, D2: irritability, D3: difficulty concentrating) joined with numbing criteria to form a factor they labeled 'dysphoria.' Two studies found that this model fit better than the DSM-based four factor model (Simms et al., 2002; Baschnagel, O'Connor, Colder, & Hawk, 2005). In contrast, the DSM-based four-factor model fit better than the dysphoria-four-factor model among individuals indirectly exposed to the September 11$^{th}$, 2001 terrorist attacks (Suvak, Maguen, Litz, Silver, & Holman, 2008), sexually harassed women (Palmieri & Fitzgerald, 2005), police officers and fire fighters involved in the 1992 air disaster in Amsterdam (Witteveen, et al., 2006), and male Vietnam veterans (Taft et al., 2007). Therefore, the DSM-IV-based four factor model, with avoidance and numbing as separate factors, is the best-supported model of the structure of PTSD.

Examining relations between PTSD and other indices of psychopathology may gloss over more complicated associations between the heterogeneous PTSD

factors and those indices (Simms, 2002). Therefore, several studies have assessed the relationships between PTSD factors and external correlates. Several relationships have been found in more than one study. The reexperiencing factor is consistently more correlated with exposure severity than are other factors (Amdur & Liberzon, 2001; Anthony, Lonigan, & Hecht, 1999; True, Rice, Eisen, & Heath, 1993). Dysphoria is usually most highly correlated with depression, and there is some evidence suggesting that dysphoria and depression may be the same construct (Simms et al., 2002; Palmieri, Weathers, Difede, & King, 2007). Hyperarousal is most strongly correlated with externalizing measures of aggression (Taft et al., 2007) and alcohol problems (Simms et al., 2002). Avoidance is generally the factor that is least correlated with most measures (Amdur & Liberzon, 2001; Palmieri et al., 2007), though it was most highly correlated with social dysfunction (Larsson, 2000).

In addition, several relationships were noteworthy in one study. Arousal was most highly correlated with worry / oversensitivity and trait anxiety (Anthony et al., 1999) and with 4 out of the 5 SCL-90 subscales tested: anxiety, depression, somatic complaints, and insufficiency (Witteveen, et al., 2006). The numbing and hyperarousal factors were more negatively correlated with life- and work-satisfaction than other factors (Palmieri & Fitzgerald, 2005).

*Genetic and Environmental Influences on the Structure of Psychopathology*

While EFAs and CFAs provide insight into the phenotypic structure of psychopathology, multivariate twin studies can provide insight regarding the extent to which this structure is influenced by genetic and environmental influences. A large twin study of the Virginia Twin Registry estimated the loadings of two common

genetic factors, two common shared environmental factors, and two common nonshared environmental factors on ten common disorders, along with disorder-specific factors (Kendler, Prescott, Myers, & Neale, 2003). This assessed which disorders shared common genetic and environmental influences. The structure of the two genetic factors matched the internalizing–externalizing structure found in phenotypic studies, with MDD, GAD, and Phobia being influenced by one genetic factor, and AD, DD, AAB, and CD being influenced by the other. There were additional disorder-specific genetic influences on AD and DD. The environmental influences didn't conform to the phenotypic structure as neatly, with one nonshared environmental factor mainly influencing MDD, GAD, and AD, and the other influencing AAB and CD only. An analysis of internalizing disorders, done separately due to computational limitations, indicated that one genetic factor influenced MDD and GAD and had a small loading on PD, and the second influenced animal and situational phobias. The environmental influences were largely disorder specific. The authors concluded that genetic factors were largely responsible for both the broad phenotypic internalizing and externalizing structure of psychopathology and the anxious-misery and fear structure of the internalizing disorders.

In addition, several studies estimated the magnitude of genetic and environmental influences on latent internalizing and externalizing factors. A study of 542 families from the Minnesota Twin Family Study, each including 17-year-old twins and both biological parents, indicated that a latent externalizing factor underlying CD, AAB, AD, and DD was highly heritable ($h^2$ = .80; Hicks, Krueger, Iacono, McGue, & Patrick, 2004). A study of 626 twin pairs found heritability

estimates of an externalizing factor underlying AAB, AD, DD, and ND at ages 17 and 24 were .70 and .66, respectively (Hicks, et al., 2007). In a sample overlapping the present study's, heritability estimates of latent internalizing (reflecting MDD, GAD, and SAD) and externalizing factors (reflecting CD, ADHD, and ODD) were .60 and .65, respectively (Cosgrove et al., 2008). Prior analyses also on this sample indicated that a latent externalizing factor underlying CD, substance experimentation, ADHD, and novelty seeking was highly heritable ($h^2$ = .81; Young, Stallings, Corley, Krauter, & Hewitt, 2000).

*Etiology of PTSD and its Comorbidity with Other Disorders*

Relatively few genetically informative studies of risk factors for exposure to potentially traumatic events and for PTSD symptoms have been conducted. Studies documented genetic influence on exposure to combat trauma (Lyons et al., 1993; True et al., 1993; Roy-Byrne et al., 2004) and noncombat assaultive trauma, but not noncombat nonassaultive trauma (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Shared environmental influences were significant for both forms of noncombat trauma. Genetic influences on exposure to traumatic experience may be mediated in part by variables such as academic performance, troublesome adolescent histories (Cordray, Polk, & Britton, 1992), conduct problems, extraversion and neuroticism (Breslau, Davis, Andreski, & Peterson, 1991), preexisting mental or substance use disorder or family history thereof (Bromet, Sonnega, & Kessler, 1998; Acierno, Resnick, Kilpatrick, Saunders, & Best, 1999; Perkonnig, Kessler, Storz, & Wittchen, 2000), all of which have been identified as risk factors for exposure to traumatic experiences.

Estimates of the heritability of PTSD, clusters of symptoms, and individual symptoms range from 21% to 41%, with little evidence for an effect of the shared environment (e.g. True et al., 1993; Stein et al., 2002; Jang et al., 2007; Chantarujikapong et al., 2001). There is conflicting evidence regarding the extent to which the genetic influences on trauma exposure are shared with those on PTSD symptoms (True et al. 1993; Stein et al., 2002).

Most studies examining the genetic and environmental influences on the comorbidity between PTSD and other psychiatric disorders were conducted on the Vietnam Era Twin Registry. Thus, the generalizability of these studies to women and noncombat trauma remain unknown. Both genetic and environmental influences appear to be important in explaining the covariation between PTSD and internalizing disorders such as GAD, PD, and DYS, with the magnitude of these influences varying across samples and disorders (Chantarujikapong et al., 2001; Koenen et al., 2003a; Koenen et al., 2003b; Koenen et al., 2008; Fu et al., 2007).

Two studies suggest that the relationship between PTSD and substance use disorders is largely explained by genetic influences (McLeod et al., 2001; Xian et al., 2000), whereas another suggests that environmental influences are more important (Koenen et al., 2006). The relationship between PTSD and CD appears to be largely environmental in nature (Fu et al., 2007; Koenen et al., 2005). Studies investigating the source of comorbidity between PTSD and general health and pain indicate that common environmental influences are the source of comorbidity, though these studies controlled for depression, which shares genetic influences with PTSD (Arguelles et al., 2006; Roy-Byrne, Noonan, Afari, Buchwald, & Goldberg, 2006).

The heterogeneity in results may be influenced by sampling error, different methods being used in different studies, and/or real differences in the etiology of the relationships between PTSD and other disorders. The heterogeneity in results may be clarified by examining the etiology of the relationship between PTSD and the latent internalizing and externalizing factors.

*The Present Study*

The present study tests whether the two factor internalizing and externalizing structure replicates in a large sample of adolescents in Colorado via EFA and CFA of eight common disorders: MDD, GAD, SAD, PTSD, CD, ODD, ADHD, and SUD. To address the conflicting findings regarding the strength of PTSD's loadings on the latent factors in previous studies, the significance of PTSD's location in the structure will be assessed by comparing the fit of three models via CFAs: one in which PTSD loads on the internalizing factor, one in which it loads on the externalizing factor, and one in which it loads on both. Given the diversity of symptoms that define PTSD and the heterogeneity of people who manifest PTSD symptoms, the relationships between each of the four DSM-based PTSD factors and the latent internalizing and externalizing factors will be assessed similarly.

In order to assess whether the phenotypic structure underlying the psychiatric disorders is explained by genetic and/or environmental influences, multivariate genetic analyses will be conducted. Cholesky models, which are the most saturated decomposition models possible, will be compared to models in which genetic and environmental influences are constrained to a smaller number of common factors based on the internalizing externalizing model of mental disorders.

The present study extends the literature in several important ways. First, it assesses the structure of psychopathology among adolescents, while most such research has been conducted with adult samples. Second, it assesses the magnitude of PTSD, ADHD, and ODD's loadings on the latent factors; preliminary studies for PTSD have yielded conflicting results (Cox et al., 2002; Slade & Watson, 2006; Kessler et al., 2005), and the only study known to the authors to include ADHD and ODD surprisingly found that both loaded much more strongly on internalizing (Kessler et al., 2005). Third, it is the first assessment of the phenotypic relationship between subfactors of PTSD and latent internalizing and externalizing factors. Finally, it tests whether the finding that genetic influences are more responsible for the structure of psychopathology than environmental influences (Kendler et al., 2003) replicates in an independent sample.

## Method

### *Participants*

Participants were 3867 adolescents assessed by the Colorado Center for Antisocial Drug Dependence. Of these, 2754 were members of twin pairs and were recruited from two community based twin samples: the Colorado Longitudinal Twin Study and the Colorado Twin Registry. Also recruited from these samples were 525 siblings of the twins. From the control sample of the Adolescent Substance Abuse Family Study, which recruits the families of adolescent patients in a treatment program for antisocial substance problems and matched control families, 338 adolescents were recruited. Two hundred and fifty adolescents were recruited from the Colorado Adoption Project. These included adoptees, their adoptive siblings,

matched controls, and matched controls' siblings. The 3867 participants came from 1729 unique families. Fifty percent of all participants were male. For genetic analyses and certain phenotypic analyses, the sample was restricted to exclude individuals for whom no sibling data was available, which reduced the sample size to 3532. To eliminate nonindependence fully, certain phenotypic analyses were conducted on a subsample with only one individual from each family (n=1729).

Written assent (from minor participants) or consent (from adult participants and guardians of minor participants) was obtained from all participants. Zygosity for same-sex twin pairs was determined via two independent processes. Interviewers completed a 9-item assessment of physical characteristics (Nichols & Bilbro, 1996), and the twin pairs' genotypes were compared at a minimum of 11 highly informative polymorphisms. Discrepancies between the interviewers' ratings and the genotype information were identified and resolved.

*Measures*

PTSD, MDD, GAD, Separation Anxiety Disorder (SAD), CD, ODD, and ADHD were assessed via the Diagnostic Interview Schedule for Children (DISC), Version 4 (Shaffer, Fisher, & Lucas, 1998), which assesses DSM-IV diagnoses (APA, 1994). Participants were assessed between 1997 and 2002. Substance abuse and dependence criteria were assessed via the Composite International Diagnostic Interview – Substance Abuse Module (CIDI-SAM; Cottler, Robins, & Helzer, 1989; Crowley, Mikulich, Ehlare, Whitmore, & MacDonald, 2001) with regard to 10 substances: tobacco, alcohol, marijuana, opioids, sedatives/hypnotics, inhalants, amphetamines, cocaine, hallucinogens, and phencyclidine. In order to maximize

endorsement rates and therefore statistical power, the criterion that symptoms cause clinically significant distress or impairment was not required for participants to be given a diagnosis. To further maximize endorsement rates, and also because older teens are largely past the age of risk for SAD and ODD, whole life diagnoses were chosen rather than prior year diagnoses. However, only prior year data was available for PTSD, therefore this variable reflects prior year symptoms and diagnoses.

As part of the PTSD module, participants were queried about their exposure to eight types of traumatic events. They frequently endorsed having seen or heard somebody getting badly hurt even though their description of the event indicated that the injury was not severe and being upset by seeing a dead body when their exposure was at a funeral. Therefore, 622 of these endorsements of exposure to traumatic experiences were examined on a case by case basis and 429 were revised to no exposure to a traumatic event, and therefore no symptoms of PTSD could be met.

*Statistical Analyses*

Data management and descriptive statistic computation were conducted using SAS 9.1. The distributions of the symptom counts were positively skewed. Therefore, the data were analyzed assuming normal continuous liability distributions underlie ordinal variables. This method retains the statistical advantages conferred by the normality assumptions for the underlying liability, retains an explicit mapping between the underlying liability and observed behavior, and correctly recovers the underlying correlations and parameter estimates (Derks, Dolan, & Boomsma, 2004; Stallings et al., 2001). A symptom sum variable for each of PTSD, MDD, GAD, SAD, CD, ODD, and ADHD was transformed into an ordinal variable with three

levels: no symptoms, symptoms endorsed but threshold for diagnosis not met, and diagnosis met. Only one substance abuse symptom is required for a diagnosis; therefore, the symptom count for substance abuse/dependence was transformed into a variable with two levels: no symptoms and one or more abuse or dependence symptoms for any of the ten substances. Similarly, symptom sums for each cluster of PTSD symptoms were transformed into ordinal variables with two levels: no symptoms met and at least one symptom. Polychoric correlations among all disorders and subfactors of PTSD were calculated. Age and gender were significant predictors of most variables, so age- and gender-specific thresholds were used when measuring correlations.

Exploratory factor analyses (EFA) and confirmatory factor analyses (CFA) of polychoric correlation matrices were conducted in Mplus (Muthén & Muthén, 1998 – 2007) and Mx (Neale et al., 2004). EFA were conducted to examine the number of factors underlying the eight psychiatric disorders in adolescents. CFA were conducted to evaluate the fit of several models. These included a one factor model, an a priori-defined two factor internalizing–externalizing model with MDD, GAD, SAD, and PTSD constituting the internalizing factor and ADHD, ODD, CD, and SUD constituting the externalizing factor, and several post-hoc models. To assess for measurement invariance across gender, EFA were conducted separately for each gender, then in CFA, we tested whether parameter estimates could be constrained across gender without a statistically significant decrease in fit.

CFA were also conducted to compare the fit of the model with PTSD loading on internalizing, the model with PTSD loading on externalizing, and the model with

PTSD loading on both internalizing and externalizing (see Figure 1). Given the observed heterogeneity in relationships between PTSD factors and other indices of distress, the relationships between each individual PTSD factor and the latent factors were similarly examined via CFA. A total of 6 subfactors were tested: reexperiencing and avoidance, both of which are identical in the DSM-based and alternative models (see Table 1), DSM-based numbing, DSM-based hyperarousal, the alternative model's dysphoria subfactor, and the alternative model's hyperarousal subfactor.

The number of factors extracted from EFAs was determined by the number of factors with eigenvalues over 1. The fits of alternative CFA models were evaluated using five indices: the chi-square statistic, the comparative fit index, the Tucker-Lewis fit index, the root mean square error of approximation (RMSEA), and the root mean square residual (RMSR). The chi-square test assesses the discrepancy between the model-estimated and sample-derived correlations, with lower values indicating better fit. The comparative fit index and the Tucker-Lewis fit index compare the model fit to a null model which assumes the latent variables in the model are uncorrelated; they vary from zero to one, with values close to one indicating good fit and values greater than .90 indicating adequate fit (Bentler, 1990). The RMSEA measures the discrepancy between the model-estimated and sample-derived correlations per degree of freedom; zero indicates perfect fit and < .05 indicates adequate fit (Browne & Cudeck, 1993). The RMSR (Jöreskog & Sörbom, 1996) indicates the average deviation of each covariance or correlation implied by the proposed model from each covariance or correlation observed in the data; lower

values indicate better fit. The RMSEA and CFI are less dependent on sample size than other fit indices (Fan, Thompson, & Wang, 1999).

To assess the structure of genetic and environmental influences on adolescent psychopathology, a Cholesky model, with eight genetic factors, eight shared environmental factors, and eight nonshared environmental factors explaining the variance and covariance among the eight variables was fitted. Next, this model was compared to a model that also contained twin-specific environmental influences and a model that contained dominant genetic influences. Then several models in which influences were constrained to two common factors according to the a priori internalizing–externalizing model were fit (e.g. Figure 2). Finally, post-hoc models based on phenotypic results and visual inspection of each influence's correlation matrices were fit.

## Results

Table 2 shows ordinal variable frequencies for each of the disorder variables, the drug use symptom variable, and the PTSD subfactor variables. For the seven disorders, 0 indicates no symptoms were met, 1 indicates at least one symptom was met but the disorder was not present, and 2 indicates that the disorder was present. For the drug use symptoms variable and the PTSD subfactors variables, 0 indicates no symptoms were met and 1 indicates one or more symptoms were met. Data were positively skewed. PTSD was relatively infrequent, with 7.8% endorsing symptoms but no disorder and 0.7% meeting criteria for the disorder. The prevalence of at least one symptom being endorsed in the PTSD subfactors range from 6.9% for

Reexperiencing and 1.2% for the alternative model hyperarousal, which contains only two items (see Table 1).

*What is the Factor Structure of Common Mental Disorders among Adolescents?*

Table 3 shows phenotypic polychoric correlations between the eight disorder variables. The mean correlation within the a priori-defined internalizing disorders of SAD, GAD, MDD, and PTSD is .36 (range .31 - .44). The mean correlation within the a priori-defined externalizing disorders of ADHD, ODD, CD, and DRUG is .41 (range .29 - .56). The mean correlation between internalizing and externalizing disorders is .27 (range from .16 to .44).

Table 4 shows phenotypic polychoric correlations by gender. Among males, the mean correlation within the a priori-defined internalizing disorders was .38, within externalizing disorders the mean was .46, and between these groups the mean was .27. Among females, the mean correlation within internalizing disorders was .35, within externalizing .39, and between internalizing and externalizing .28. These patterns are generally similar for males and females, with females having somewhat lower correlations within externalizing disorders.

An exploratory factor analysis of the eight disorder variables yielded two factors with eigenvalues greater than one. The two factor solution fit moderately well ($\chi^2$ = 483.70, CFI = .93, TLI = .86, RMSEA = .10, Standardized RMSR = .04), but the three factor solution fit substantially better ($\chi^2$ = 92.35, CFI = .99, TLI = .95, RMSEA = .06, Standardized RMSR = .02). Table 5 shows factor loadings for the two- and three-factor solutions. As expected, in the two factor solution SAD, GAD, MDD, and PTSD have substantial loadings on one factor and ADHD, ODD, CD, and DRUG

have substantial loadings on the other. The two factors have a positive correlation of .52. Interestingly, ADHD and ODD have substantial cross loadings such that they load more strongly on internalizing. A different pattern is evident in the three factor solution. Here, SAD, GAD, MDD, and PTSD load strongly on the first factor, DRUG and to a much lesser extend CD load on the second factor, and ADHD, ODD, and CD load strongly on the third factor. The cross loadings are noticeably lower in the three factor solution. The first and third factors correlate more strongly (.58) than do the first and second (.13) and the second and third (.23), suggesting that ADHD, ODD, and CD have higher correlations with internalizing than DRUG does, and that DRUG has lower correlations with both the internalizing factor and the ADHD/ODD/CD factor. Since DRUG was relatively independent of other factors, an EFA was run excluding DRUG. This analysis yielded a two factor solution conforming to the internalizing–externalizing model with relatively low cross loadings. Table 5 shows the factor loadings from the EFA.

Table 6 shows factor loadings for exploratory analyses for males and females separately, both in the full sample (n=3867) and in the sample with only one individual per family (n = 1729). In the full sample, males adhere to the internalizing–externalizing model, with only ADHD having a large cross loading on internalizing. Among females, all disorders load primarily on one factor except CD and DRUG, which load primarily on the other. Among the sample with only one individual per family, the pattern is similar for females. However, the pattern is less clear for males. Significant cross loadings exist for MDD, PTSD, and ADHD.

Table 7 shows the fit of various models assessed via confirmatory factor analysis. The a priori internalizing–externalizing model fit significantly better that a one factor model. A two factor model in which ADHD and ODD loaded on the internalizing factor only fit better than the a priori model. Allowing ADHD and ODD to load on both factors further improved fit. A three factor model based on the three factor EFA solution in which the a priori internalizing disorders load on one factor, ADHD, ODD, and CD load on a second factor, and CD and DRUG load on a third factor was also fit. CD was included on the third factor due to its small but statistically significant loading in the EFA and because the statistical program M+ cannot fit a model with only observed variable loading on a latent factor. The three factor model fit significantly better than any of the two factor models.

Each of the models displayed in table 8 were fit both with parameters allowed to vary across genders and with parameters constrained to be equal across genders. Even though these analyses were conducted on the smaller sample with only one member from each family included, parameter estimates could not be constrained across genders without a significant decrease in model fit for each of the five models.

*On Which Factors and How Strongly Do ..PTSD and its Subfactors Load?*

Table 9 shows the correlations between the six PTSD subfactors and the seven other psychiatric disorders and each subfactor's average correlation with the three other internalizing disorders and with the four externalizing disorders. There was little variance across correlations, with most correlations being between .2 and .4. Most subfactors were only slightly more highly correlated with internalizing disorders

than with externalizing disorders. This difference was somewhat greater for the DSM – Numbing subfactor and the Alternative Model's Hyperarousal subfactor.

Table 10 shows model fits and the strength of factor loadings in models with PTSD and each subfactor loading on internalizing only, externalizing only, and both latent factors. PTSD loaded strongly on internalizing and not at all on externalizing. However, it is worth noting that the three factor EFA solution did include a sizeable loading of PTSD on the ADHD/ODD/CD factor (.28). Each subfactor loaded more strongly on internalizing than on externalizing. In the models with subfactors loading on internalizing only, there was little variance in the strength of loadings across subfactors, with a range of .56 to .62. In contrast, in the models with subfactors loading on internalizing and externalizing, there was more variance in the strength of loadings on externalizing, with a range of -.02 to .24. Therefore, for some subfactors, the best fitting model had the subfactor loading on internalizing only, whereas for others, the best fitting model had the subfactor loading on both internalizing and externalizing.

*What is the Structure of Genetic and Environmental Influences on Common Mental Disorders among Adolescents?*

Table 11 shows the within-trait, cross-sibling correlations. Since MZ twins share greater genetic similarity than do DZ twins and full sibling pairs, greater MZ correlations suggest the presence of genetic influences. In this sample, MZ correlations tend to be greater than DZ and full sibling correlations, which is consistent with a role of genetics in the etiology of these disorders. DZ correlations are generally not much greater than half the MZ correlations, which suggests that

shared environmental influences are small. Similarly, DZ correlations are generally not much greater than full sibling correlations, suggesting that the twin specific environmental influences are small. The correlation for MZ twins, who share both 100% of their segregating genes and the shared environment, are well below 1, suggesting a substantial role of the nonshared environment.

Table 12 shows cross-trait, cross-sibling correlations by sibling type. MZ twins' cross trait correlations tend to be higher than DZ and full sib correlations, suggesting that common genetic influences contribute to the phenotypic correlations. The MZ cross trait correlations are generally lower than the phenotypic correlations, suggesting that common nonshared environmental influences and/or common measurement error also contribute to the phenotypic correlations.

Table 13 shows the fit of the biometrical genetic models tested. The ACE model evinced a better combination of fit and parsimony as indicated by a lower Akaike's Information Criterion (AIC) than both the ACTE model and the ADE model. Therefore, the ACE Cholesky model was selected as the base model for further comparisons. First, we tested whether genetic influences, shared environmental influences, and nonshared environmental influences could be constrained to two common factors according to the a priori internalizing–externalizing model. The model with genetic influences constrained to two latent factors resulted in a statistically significant decrease in model fit. In contrast, shared environmental influences, which were generally small (see Table 13), could be constrained to one common factor; the restricted model led to a slight decrease in AIC relative to the ACE Cholesky model, indicating a slightly better combination of fit and parsimony.

Similar to genetic influences, nonshared environmental influences could not be constrained to two latent factors without a statistically significant decrease in model fit.

Next, the fit of two post hoc models with a smaller number of genetic factors than that in the Cholesky model was assessed. In each, shared environmental influences and nonshared environmental influences followed the Cholesky model. First, a model that was inspired by the exploratory factor analyses results was fit; the first common genetic factor loaded on internalizing disorders, the second loaded on ADHD, ODD, and CD, and the third loaded on CD and DRUG. This model exhibited a less desirable combination of fit and parsimony, as indicated by the AIC.

In the next model, the genetic factors were constrained following a visual inspection of the correlation matrix of genetic influences on the eight disorders derived from the Cholesky model. In this model, the first genetic factor loaded on all disorders except DRUG, the second loaded on PTSD and the four externalizing disorders, the third loaded on CD and DRUG, and only DRUG had disorder-specific genetic influences. This model evinced a lower AIC than the Cholesky model. Neither shared nor nonshared environmental influences could be constrained in models with this structure of genetic influences. Interestingly, although the phenotypic model indicated that PTSD loaded much more strongly on internalizing than on externalizing, the path from the common genetic factor loading on externalizing disorders to PTSD could not be dropped from the model, as the correlation between genetic influences on PTSD and genetic influences on ADHD

and ODD was very high. In contrast, the correlation between genetic influences on PTSD and genetic influences on CD and DRUG were much lower.

One-fifth to one-quarter of the variance in each disorder was due to genetic influences, with the exception of CD and DRUG, which had higher heritabilities (Table 14). Shared environmental influences were generally small, and nonshared environmental influences were large.

The proportion of covariance due to A, C, and E is shown for each disorder pair in Table 15. On average, about half of the covariance between disorders is due to shared genetic influences (mean = .50) and approximately one-third is due to common nonshared environmental influences (mean = .31). Slightly more is due to shared genetic influences within internalizing disorders and within externalizing disorders (both means = .55) than between internalizing and externalizing disorders (mean = .47).

Path coefficients for the final genetic model are shown in table 16. The table shows moderate loadings of three common genetic factors and one DRUG specific genetic path. Paths in the shared environmental matrix tended to be low. The nonshared environmental matrix shows large loadings on the diagonal (contributing to disorders' variance) and small loadings off the diagonal (contributing to covariance between disorders).

## Discussion

The goal of the present study was to assess: 1) whether the two-factor structure of common psychiatric disorders found among adults (e.g. Krueger, et al., 1998) replicates among adolescents; 2) the strength and the significance of PTSD, ADHD,

and ODD's loadings on latent factors; 3) the strength and significance of PTSD subfactors' loadings on latent internalizing and externalizing factors; 4) the latent structure of genetic and environmental influences on common psychiatric disorders among adolescents.

*The Factor Structure of Psychopathology among Adolescents*

Exploratory and confirmatory factor analysis results indicated that a two factor model of psychopathology fit the data adequately. In the exploratory factor analysis, in which two eigenvalues greater than one were extracted, disorders generally segregated as expected according to the internalizing–externalizing model of psychopathology, with SAD, GAD, MDD, and PTSD loading primarily on one factor, and ADHD, ODD, CD, and DRUG loading on another. However, ADHD and ODD had significant cross loadings on internalizing that were slightly greater than their loadings on externalizing. Cross loadings were generally lower in the three factor solution, as DRUG and to a much lesser extent CD loaded on one latent factor and ADHD, ODD, and CD loaded on another. Confirmatory factor analysis results revealed that the two factor internalizing–externalizing model fit better than a one factor model. A model in which ADHD and ODD loaded on internalizing only fit better, as did models allowing them to load on both factors.

In both exploratory and confirmatory factor analyses, a three factor model with two externalizing factors fit better than any of the two factor models. In this model, ADHD, ODD, and CD formed one externalizing factor, and CD and DRUG formed another externalizing factor. In the EFA, the ADHD/ODD/CD factor was more highly correlated with internalizing (.58) than with the CD/DRUG factor (.23).

The present results among adolescents are consistent with prior research documenting that a small number of latent factors underlie the observed patterns of covariance among common disorders (Krueger et al., 1998; Krueger 1999; Vollebergh et al., 2001; Krueger, McGue, Iacono, 2001; Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003). However, the present results are inconsistent with prior research in that it identified two externalizing factors and that one of these factors (ADHD/ODD/CD) correlated more highly with internalizing than the other externalizing factor (CD/DRUG).

Several hypotheses may explain this finding. This study was different from prior studies in that it was conducted on adolescents rather than adults and included disorders that have generally not been included in prior analyses. Therefore it is impossible to discern to what extent each difference influenced the results.

It may be the case that ODD shares features of internalizing disorders such as negative affectivity as well as features of externalizing disorders such as aggression and disinhibition. That ODD is related to both internalizing and externalizing is recognized by authors of DSM, as ODD is defined such that in order to meet criteria for a diagnosis, an individual must not meet criteria for conduct disorder and symptoms must not exclusively occur during the course of a mood disorder. Similarly, ADHD may share features of internalizing disorders, as features such as difficulty concentrating are common among internalizing disorders. It may be revealing to analyze the hyperactive and inattentive subtypes of ADHD separately, as hyperactivity may be more associated with externalizing and inattention may be relatively more associated with internalizing (Lahey, Schaughency, Hynd, Carlson, &

Nieves, 1987; Lahey et al., 1984; Lahey et al., 1985; Barkley et al., 1990). Another hypothesis explaining the present study's results is that unlike ADHD, ODD, and CD, drug use disorders require a certain environmental context to develop, namely exposure to drug use opportunities.

Future studies should assess whether these finding replicate in other adolescent samples. If future studies corroborate the current findings, the results would suggest that while CD is known to be a risk factor for adult substance use disorders, ADHD and ODD are less indicative of vulnerability to adult drug use disorders.

Interesting gender differences may exist in the structure of psychopathology among adolescents. In the two factor solution, ADHD and ODD load more strongly on externalizing among males, although the difference for ADHD is small. In contrast, ADHD and ODD load more strongly on internalizing among females. Model fitting in Mx showed that for a variety of models, parameter estimates could not be constrained across genders without a significant decrease in model fit, suggesting that the difference in the factor structures between males and females may be meaningful. However, when parsing these variables by gender, certain cells in the multivariate cross frequency tables are likely infrequent, possibly leading to differing results across genders that may be spurious. The gender differences found in the present study need to be corroborated by future studies including larger samples.

*The Strength and Significance of PTSD and its Subfactors' Loadings on Latent Factors*

PTSD loaded strongly on the internalizing factor and insignificantly on the externalizing factor in CFAs. Including the present study, PTSD has now loaded as strongly on the internalizing factor as the more prototypical internalizing disorders in three out of four studies. However, the three factor EFA solution indicated that PTSD had a sizeable loading on the ADHD/ODD/CD factor. Each of the PTSD subfactors loaded more strongly on internalizing than on externalizing. Further, there was little variance in the magnitude of subfactors' loading on internalizing, with a relatively small range of from .56 to .62. However, in models where the subfactor is free to load on both internalizing and externalizing, there is variance in the magnitude and significance of loadings on externalizing. The loading on externalizing is statistically significant for Reexperiencing, Avoidance, DSM – Hyperarousal, and the alternative model's Dysphoria subfactor, with a range in magnitude from .15 to .24. In contrast, DSM – Numbing and the alternative model's Hyperarousal.subfactor do not load significantly on externalizing. The consistency in results across subfactors suggests that PTSD is a relatively unidimensional disorder in this sample. That is, although PTSD has consistently been found to be a multifactorial disorder with four factor solutions being most common among adults, it may be a unidimensional construct in this sample of adolescents. Therefore, future studies in which PTSD is demonstrated to be multidimensional should examine the heterogeneity across subfactors in relationships with latent internalizing and externalizing factors.

*Structure of Genetic and Environmental Influences on Common Mental Disorders*

Fitting multivariate twin models indicated that a Cholesky model with eight genetic, eight shared environmental, and eight nonshared environmental factors fit

better than models with either nonadditive genetic influences or twin-specific environmental influences. Genetic and nonshared environmental factors could not be constrained to two factors without a statistically significant decrease in fit. In contrast, constraining shared environmental influences, which were generally small, to one common factor improved the combination of model fit and parsimony.

In one post-hoc model, genetic influences were constrained to three factors according to the phenotypic exploratory factor analysis results, with one common factor loading on internalizing, one common factor loading on ADHD, ODD, and CD, and another loading on CD and DRUG. This model fit significantly worse than the Cholesky model.

A second post hoc model was derived from visual inspection of the Cholesky model's correlation matrix of genetic influences and contained one common factor loading on all disorders except DRUG, another on PTSD and the four a priori-defined externalizing disorders, a third on CD and DRUG, and a fourth factor reflecting specific genetic influences on DRUG. This model improved the combination of fit and parsimony over the Cholesky model. Interestingly, the correlation between genetic influences on PTSD and genetic influences on ADHD and ODD was very high and the correlation between genetic influences on PTSD and genetic influences on CD and DRUG were much lower. This indicates that relatively more of the comorbidity between PTSD and ADHD and ODD is due to genetic influences, and relatively more of the comorbidity between PTSD and CD and DRUG are due to environmental influences.

These findings are somewhat inconsistent with prior research (Kendler et al., 2003), which found that genetic influences on common mental disorders among adults could be constrained to two factors dictated by the internalizing–externalizing model of psychopathology. This inconsistency may be due to the different set of disorders included in each study. Specifically, Kendler et al. included 'phobia', which falls under the 'fear' subfactor of internalizing, and did not include PTSD and the childhood diagnoses of ADHD and ODD. The inclusion of PTSD is noteworthy as the genetic influences on PTSD were highly correlated with those on ADHD and ODD. Similarly, the inclusion of ADHD and ODD is significant, as these disorders separated from the internalizing and DRUG factors in the phenotypic analyses in the present study, and the internalizing and DRUG factors match the ones found in Kendler et al. quite closely.

Alternatively, the inconsistency may be attributable to the different ages of the samples, with the present study being conducted on adolescents and Kendler et al. being conducted on adults. This would suggest that the structure of genetic and environmental influences on common mental disorders is different among adults and adolescents. While Kendler et al (2003) may be cited to support focusing psychiatric genetic research on latent internalizing and externalizing constructs, the present study suggests that the structure of genetic and environmental influences on common adolescent disorders may be more complicated.

The present study has several strengths. It was conducted on a large population based sample and used gold standard assessment methods to obtain DSM diagnoses. It is only the second study of its kind known to the authors to include ADHD and

ODD, and the first to include subfactors of the multifactorial disorder PTSD. This study is also only the second study known to the authors to examine the structure of genetic and environmental influences on a large set of common mental disorders, and it was the first to do so among adolescents.

Several limitations of the present study are also noteworthy. The sample contained related individuals, necessitating adjustments to be made for non-independence. Also, endorsement rates for some symptoms and disorders, such as PTSD and its subfactors, were low. The low endorsement rates for PTSD symptoms meant that we could not examine the factor structure of PTSD symptoms in the present sample. In order to maximize endorsement rates and therefore statistical power, participants were not required to evince clinically significant distress or impairment to be given a diagnosis, as is required by the DSM. While whole-life diagnoses were used for all other variables, only prior year diagnoses were available PTSD. Further, the reliability of the DISC in assessing PTSD is unknown. The diagnostic definitions used for CD and ADHD did not conform strictly to DSM definitions with regard to clustering or age of onset of symptoms.

The finding that the broad two factor, internalizing–externalizing structure of psychopathology supported among adults replicated among adolescents is consistent with calls for a dimensional psychopathological nosology (e.g. Clark 2005; Watson 2005). The present study suggests that a dimensional nosology based on the internalizing externalizing model of psychopathology would be reasonable for adolescents. The similarity in structure between mood, anxiety, antisocial behavior, and substance use disorders among adults and adolescents suggests that it may be

appropriate to remove the nosological distinction between adolescent and adult psychopathology. However, with the inclusion of ADHD and ODD, two externalizing factors may be necessary. The present study suggests that PTSD fits into the internalizing–externalizing model as well as the more prototypical internalizing disorders such as MDD and GAD. Fewer latent factors may underlie patterns of covariance among PTSD symptoms among adolescents than adults, though this should be corroborated in larger samples and samples with greater prevalence of PTSD symptoms. The present study refutes prior research suggesting that genetic influences largely determine the internalizing–externalizing structure of psychopathology and that nonshared environmental influences largely determine risk for specific disorders within internalizing or externalizing, at least in this adolescent sample.

# References

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin, 85*(6), 1275-1301.

Acierno, R., Resnick, H., Kilpatrick, D. G., Saunders, B., & Best, C. L. (1999). Risk factors for rape, physical assault, and posttraumatic stress disorder in women: Examination of differential multivariate relationships. *Journal of Anxiety Disorders, 13*(6), 541-563.

Amdur, R. L., & Liberzon, I. (2001). The structure of posttraumatic stress disorder symptoms in combat veterans: A confirmatory factor analysis of the impact of event scale. *Journal of Anxiety Disorders, 15*(4), 345-357.

American Psychiatric Association. *Diagnostic and statistical manual of mental disorders (3rd ed, revised.).* Washington, D.C.: American Psychiatric Association; 1987.

American Psychiatric Association. *Diagnostic and statistical manual of mental disorders (4th ed.).* Washington, D.C.: American Psychiatric Association; 1994.

Anthony, J. L., Lonigan, C. J., & Hecht, S. A. (1999). Dimensionality of posttraumatic stress disorder symptoms in children exposed to disaster: Results

from confirmatory factor analyses. *Journal of Abnormal Psychology, 108*(2), 326-336.

Arguelles, L. M., Afari, N., Buchwald, D. S., Clauw, D. J., Furner, S., & Goldberg, J. (2006). A twin study of posttraumatic stress disorder symptoms and chronic widespread pain. *Pain, 124*(1-2), 150-157.

Asmundson, G. J. G., Frombach, I., McQuaid, J., Pedrelli, P., Lenox, R., & Stein, M. B. (2000). Dimensionality of posttraumatic stress symptoms: A confirmatory factor analysis of DSM-IV symptom clusters and other symptom models. *Behaviour Research and Therapy, 38*(2), 203-214.

Asmundson, G. J. G., Stapleton, J. A., & Taylor, S. (2004). Are avoidance and numbing distinct PTSD symptom clusters? *Journal of Traumatic Stress, 17*(6), 467-475.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107,* 238-246.

Asmundson, G. J. G., Stein, M. B., & McCreary, D. R. (2002). Posttraumatic stress disorder symptoms influence health status of deployed peacekeepers and nondeployed military personnel. *Journal of Nervous and Mental Disease, 190*(12), 807-815.

Barkley, R. A., DuPaul, G. J., & McMurray, M. B. (1990). Comprehensive

evaluation of attention deficit disorder with and without hyperactivity as defined by research criteria. *Journal of Consulting and Clinical Psychology, 58* (6), 775-789.

Baschnagel, J. S., O'Connor, R. M., Colder, C. R., & Hawk, L. W., Jr. (2005). Factor structure of posttraumatic stress among western new york undergraduates following the september 11th terrorist attack on the world trade center. *Journal of Traumatic Stress, 18*(6), 677-684.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238-246.

Blanchard, E. B., Hickling, E. J., Taylor, A. E., & Loos, W. R. (1994). Psychological morbidity associated with motor vehicle accidents. *Behaviour Research and Therapy, 32*(3), 283-290.

Breslau, N., Davis, G. C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry, 48*(3), 216-222.

Bromet, E., Sonnega, A., Kessler, R.C. (1998). Risk factors for DSM-III-R posttraumatic stress disorder: Findings from the national comorbidity survey. *American Journal of Epidemiology, 147*(4):353-361.

Browne, M. W., & Cudeck, R. (1987). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), Testing structural equation models (pp. 136-162). Newbury Park, CA: Sage Publications, Inc.

Buckley, T. C., Blanchard, E. B., & Hickling, E. J. (1998). A confirmatory factor analysis of posttraumatic stress symptoms. *Behaviour Research and Therapy, 36*(11), 1091-1099.

Chantarujikapong, S. I., Scherrer, J. F., Xian, H., Eisen, S. A., Lyons, M. J., Goldberg, J., et al. (2001). A twin study of generalized anxiety disorder symptoms, panic disorder symptoms and post-traumatic stress disorder in men. *Psychiatry Research, 103*(2-3), 133-146.

Clark, L. A. (2005). Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology.Special Issue: Toward a Dimensionally Based Taxonomy of Psychopathology, 114*(4), 505-521.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology.Special Issue: Diagnoses, Dimensions, and DSM-IV: -n the Science of Classification, 100*(3), 316-336.

Comptom, W.M., Cottler, L.B., Dorsey, K.B., Spitznagel, E.L., & Mager, D.E., (1996). Comparing assessments of *DSM-IV* substance dependence disorders

using CIDI-SAM: a comprehensive substance abuse interview. *British Journal of Addiction, 84*:801-814.

Cordray, S. M., Polk, K. R., & Britton, B. M. (1992). Premilitary antecedents of post-traumatic stress disorder in an oregon cohort. *Journal of Clinical Psychology*, *48*(3), 271-280.

Cosgrove, V.E., Rhee, S.H., Gelhorn, H., Boeldt, D., Corley, R.C., Ehringer, M.A., Young, S.E., & Hewitt, J.K., (2008). Covariance between internalizing and externalizing disorders in adolescents. Manuscript in preparation.

Cottler, L.B., Ronibs, L.N., & Helzer, J.E., (1989). The reliability of the CIDI-SAM: A comprehensive substance abuse interview. *British Journal of Addiciton*, *84*:801-814.

Crowley, T.J., Mikulich, S.K., Ehlare, K.M., Whitmore, E.A., & MacDonald, M.J., (2001). Validity of structured clinical evaluations in adolescents with conduct and substance problems. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*:265-273.

Cox, B.J., Clara, I.P., Enns, M.W., (2002). Posttraumatic stress disorder and the structure of common mental disorders. *Depression and anxiety*, 15(4):168-171.

Davidson, J., Foa, E.B., Blank, A.S., Brett, E.A., Fairbank, J., Green, B.L., et al., (1994). Posttraumatic stress disorder. In T.A. Widiger, A.J. Frances, H.A. Pincus,

R. Ross, M.B. First, & W.W. Davis (Eds.), *DSM-IV Sourcebook, Vol. 2* (pp. 577-605).

Derks, E.M., Dolan, C.V., & Boomsma, D.I., (2004). Effects of censoring on parameter estimates and power ingenetic modeling. *Twin Research, 7*(6):659-669.

Fan, X., Thompson, B., & Wang, L. (1999). Effects of sample size, estimation method, and model specification on structural equation modeling fit indexes. *Structural Equation Modeling, 6,* 56-83.

Felmingham, K. L., Bryant, R. A., Kendall, C., & Gordon, E. (2002). Event-related potential dysfunction in posttraumatic stress disorder: The role of numbing. *Psychiatry Research, 109*(2), 171-179.

Foa, E. B., Zinbarg, R., & Rothbaum, B. O. (1992). Uncontrollability and unpredictability in post-traumatic stress disorder: An animal model. *Psychological Bulletin, 112*(2), 218-238.

Fu, Q., Koenen, K. C., Miller, M. W., Heath, A. C., Bucholz, K. K., Lyons, M. J., et al. (2007). Differential etiology of posttraumatic stress disorder with conduct disorder and major depression in male veterans. *Biological Psychiatry, 62*(10), 1088-1094.

Hicks, B. M., Blonigen, D. M., Kramer, M. D., Krueger, R. F., Patrick, C. J., Iacono, W. G., et al. (2007). Gender differences and developmental change in

externalizing disorders from late adolescence to early adulthood: A longitudinal twin study. *Journal of Abnormal Psychology, 116*(3), 433-447.

Hicks, B. M., Krueger, R. F., lacono, W. G., McGue, M., & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders: A twin-family study. *Archives of General Psychiatry, 61*(9), 922-928.

Horney, K. (1945). *Our inner conflicts; a constructive theory of neurosis*. New York, NY, US: W W Norton & Co.

Jang, K. L., Taylor, S., Stein, M. B., & Yamagata, S. (2007). Trauma exposure and stress response: Exploration of mechanisms of cause and effect. *Twin Research and Human Genetics, 10*(4), 564-572.

Jensen, P., Roper, M., Fisher, P., Piacentini, J., Canino, G., et al. (1995). Test-retest reliability of the Diagnostic Interview Schedule for Children (DISC 2.1). *Archives of General Psychiatry*, 52, 61-71.

Jöreskog, K. G. & Sörbom, D. (1996). LISREL 8: User's Reference Guide. Chicago, IL: Scientific Software International.

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry, 60*(9), 929-937.

Kessler, R.C., Chiu, W.T., Demler, O., Merikangas, K.R., Walters, E.E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry,* 62(6):617-627.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., et al. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry, 52*(12), 1048-1060.

King, D. W., Leskin, G. A., King, L. A., & Weathers, F. W. (1998). Confirmatory factor analysis of the clinician-administered PTSD scale: Evidence for the dimensionality of posttraumatic stress disorder. *Psychological Assessment, 10*(2), 90-96.

Koenen, K. C., Fu, Q. J., Ertel, K., Lyons, M. J., Eisen, S. A., True, W. R., et al. (2008). Common genetic liability to major depression and posttraumatic stress disorder in men. *Journal of Affective Disorders, 105*(1-3), 109-115.

Koenen, K. C., Hitsman, B., Lyons, M. J., Niaura, R., McCaffery, J., Goldberg, J., et al. (2005). A twin registry study of the relationship between posttraumatic stress disorder and nicotine dependence in men. *Archives of General Psychiatry, 62*(11), 1258-1265.

Koenen, K. C., Hitsman, B., Lyons, M. J., Stroud, L., Niaura, R., McCaffery, J., et al. (2006). Posttraumatic stress disorder and late-onset smoking in the vietnam era twin registry. *Journal of Consulting and Clinical Psychology, 74*(1), 186-190.

Koenen, K. C., Lyons, M. J., Goldberg, J., Simpson, J., Williams, W. M., Toomey, R., et al. (2003a). Co-twin control study of relationships among combat exposure, combat-related PTSD, and other mental disorders. *Journal of Traumatic Stress, 16*(5), 433-438.

Koenen, K. C., Lyons, M. J., Goldberg, J., Simpson, J., Williams, W. M., Toomey, R., et al. (2003b). A high risk twin study of combat-related PTSD comorbidity. *Twin Research, 6*(3), 218-226.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56*(10), 921-926.

Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (DSM-III-R): A longitudinal-epidemiological study. *Journal of Abnormal Psychology, 107*(2), 216-227.

Krueger, R. F., Chentsova-Dutton, Y. E., Markon, K. E., Goldberg, D., & Ormel, J. (2003). A cross-cultural study of the structure of comorbidity among common psychopathological syndromes in the general health care setting. *Journal of Abnormal Psychology, 112*(3), 437-447.

Krueger, R. F., Markon, K. E., Patrick, C. J., & Iacono, W. G. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *Journal of Abnormal Psychology.Special Issue: Toward a Dimensionally Based Taxonomy of Psychopathology, 114*(4), 537-550.

Krueger, R. F., McGue, M., & Iacono, W. G. (2001). The higher-order structure of common DSM mental disorders: Internalization, externalization, and their connections to personality. *Personality and Individual Differences, 30*(7), 1245-1259.

Lahey, B. B., Schaughency, E. A., Frame, C. L., & Strauss, C. C. (1985). Teacher ratings of attention problems in children experimentally classified as exhibiting attention deficit disorder with and without hyperactivity. Journal of the American Academy of Child Psychiatry, 24 (5), 613-616.

Lahey, B. B., Schaughency, E. A., Hynd, G. W., Carlson, C. L., & Nieves, N. (1987). Attention deficit disorder with and without hyperactivity: Comparison of behavioral characteristics of clinic-referred children. Journal of the American Academy of Child and Adolescent Psychiatry, 26 (5), 718-723.

Lahey, B. B., Schaughency, E. A., Strauss, C. C., & Frame, C. L. (1984). Are attention deficit disorders with and without hyperactivity similar or dissimilar disorders? Journal of the American Academy of Child Psychiatry, 23 (3), 302-309.

Larsson, G. (2000). Dimensional analysis of the impact of event scale using structural equation modeling. *Journal of Traumatic Stress, 13*(2), 193-204.

Little, R.J.A. & Rubin, D.B. (2002). *Statistical Analysis with Missing Data*, 2nd edition, New York: John Wiley.

Lyons, M.J., Goldberg, J., Eisen, S.A., True, W., Tsuang, M.T., Myer, J.M., Henderson, W.G. (1993). Do genes influence exposure to trauma? A twin study of combat. *American Journal of Medical genetics (Neuropsychiatric Genetics)*, 48:22-27.

McLeod, D. S., Koenen, K. C., Meyer, J. M., Lyons, M. J., Eisen, S., True, W., et al. (2001). Genetic and environmental influences on the relationship among combat exposure, posttraumatic stress disorder symptoms, and alcohol use. *Journal of Traumatic Stress, 14*(2), 259-275.

Miller, M. W., Kaloupek, D. G., Dillon, A. L., & Keane, T. M. (2004). Externalizing and internalizing subtypes of combat-related PTSD: A replication and extension using the PSY-5 scales. *Journal of Abnormal Psychology, 113*(4), 636-645.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology.Special Issue: Diagnoses, Dimensions, and DSM-IV: -n the Science of Classification, 100*(3), 245-261.

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49*, 377-412.

Muthén, L.K. & Muthén, B.O. (1998-2007). Mplus User's Guide, Fifth Edition. Los Angeles: Muthén & Muthén.

Neale, M.C., Boker, S.M., Xie, G., & Maes, H.H (2004). Mx: Statistical Modeling, Sixth Edition. Virginia Commonwealth University, Department of Psyhiatry, Richmond.

Nichols, R.C., Bilbro, W.C. (1996). The diagnosis of twin zygosity. *Acta Genet Stat Med, 16*:265-275.

Palmieri, P. A., & Fitzgerald, L. F. (2005). Confirmatory factor analysis of posttraumatic stress symptoms in sexually harassed women. *Journal of Traumatic Stress, 18*(6), 657-666.

Palmieri, P.A., Weathers, F.W., Difede, J., & King, D.W. (2007). Confirmatory factor analysis of the PTSD checklist and the clinican-administered PTSD scale in disaster workers exposed to the world trade center ground zero. *Journal of Abnormal Psychology, 116*(2), 329-341.

Perkonigg, A., Kessler, R. C., Storz, S., & Wittchen, H. (2000). Traumatic events and post-traumatic stress disorder in the community: Prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavica, 101*(1), 46-59.

Roy-Byrne, P., Arguelles, L., Vitek, M. E., Goldberg, J., Keane, T. M., True, W. R., et al. (2004). Persistence and change of PTSD symptomatology: A longitudinal co-twin control analysis of the vietnam era twin registry. *Social Psychiatry and Psychiatric Epidemiology, 39*(9), 681-685.

Roy-Byrne, P. P., Noonan, C., Afari, N., Buchwald, D., & Goldberg, J. (2006). Is the association between posttraumatic stress disorder symptoms and poor health due to a common familial or genetic factor? *General Hospital Psychiatry, 28*(5), 408-413.

Ruscio, A. M., Ruscio, J., & Keane, T. M. (2002). The latent structure of posttraumatic stress disorder: A taxometric investigation of reactions to extreme stress. *Journal of Abnormal Psychology, 111*(2), 290-301.

Shaffer D, Fisher P, Lucas C, The NIMH DISC Editorial Board (1998), Diagnostic Interview Schedule for Children, Version IV. New York: Columbia University, Division of Psychiatry.

Shaffer, D., Schwab-Stone, M., Regier, D., Fisher, P., Cohen, P., Piacentini, J., et al. (1991). Revised versions of the Diagnostic Interview Schedule for Children (DISC): Preparation, field testing, inter-rater, reliability and acceptability. Unpublished manuscript.

Simms, L. J., & Watson, D., (1999, August). *Exploratory factor analysis of PTSD symptoms in two military samples.* Poster presented at the 107th Annual Convention of the American Psychological Association.

Simms, L. J., Watson, D., & Doebbelling, B. N. (2002). Confirmatory factor analyses of posttraumatic stress symptoms in deployed and nondeployed veterans of the gulf war. *Journal of Abnormal Psychology, 111*(4), 637-647.

Slade, T., & Watson, D. (2006). The structure of common DSM-IV and ICD-10 mental disorders in the australian general population. *Psychological Medicine, 36*(11), 1593-1600.

Stallings, M.C., Hewitt, J.K., Lessem, J.M., Young, S.E., Corley, R.P., Mikulich, S.K., & Crowley, T.J. (2001). Modeling the familial transmission of alcohol dependence symptom counts in clinical and control family pedigrees [Abstract]. *Behavior Genetics, 31,* 470.

Stein, M. B., Jang, K. L., Taylor, S., Vernon, P. A., & Livesley, W. J. (2002). Genetic and environmental influences on trauma exposure and posttraumatic stress disorder symptoms: A twin study. *American Journal of Psychiatry, 159*(10), 1675-1681.

Suvak, M., Maguen, S., Litz, B. T., Silver, R. C., & Holman, E. A. (2008). Indirect exposure to the september 11 terrorist attacks: Does symptom structure resemble PTSD? *Journal of Traumatic Stress, 21*(1), 30-39.

Taft, C. T., Kaloupek, D. G., Schumm, J. A., Marshall, A. D., Panuzio, J., King, D. W., et al. (2007). Posttraumatic stress disorder symptoms, physiological reactivity, alcohol problems, and aggression among military veterans. *Journal of Abnormal Psychology, 116*(3), 498-507.

Taylor, S., Kuch, K., Koch, W. J., Crockett, D. J., & Passey, G. (1998). The structure of posttraumatic stress symptoms. *Journal of Abnormal Psychology, 107*(1), 154-160.

Taylor, S., Fedoroff, I. C., Koch, W. J., Thordarson, D. S., Fecteau, G., & Nicki, R. M. (2001). Posttraumatic stress disorder arising after road traffic collisions: Patterns of response to cognitive-behavior therapy. *Journal of Consulting and Clinical Psychology, 69*(3), 541-551.

True, W. R., Rice, J., Eisen, S. A., & Heath, A. C. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry, 50*(4), 257-264.

Vollebergh, W.A., Iedema, J., Bijl, R.V., de Graaf, R., Smit, F., Ormel, J. (2001). The structure and stability of common mental disorders: the NEMESIS study. *Archives of General Psychiatry,* 58(6):597-603.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology.Special Issue: Toward a Dimensionally Based Taxonomy of Psychopathology, 114*(4), 522-536.

Witteveen, A. B., Van der Ploeg, E., Bramsen, I., Huizink, A. C., Slottje, P., Smid, T., et al. (2006). Dimensionality of the posttraumatic stress response among police officers and fire fighters: An evaluation of two self-report scales. *Psychiatry Research, 141*(2), 213-228.

World Health Organization (1989). *Composite International Diagnostic Interview.* Geneva, Switzerland: World Health Organization, Division of Mental Health.

Xian, H., Chantarujikapong, S. I., Scherrer, J. F., Eisen, S. A., Lyons, M. J., Goldberg, J., et al. (2000). Genetic and environmental influences on posttraumatic stress disorder, alcohol and drug dependence in twin pairs. *Drug and Alcohol Dependence, 61*(1), 95-102.

Young, S.E., Stallings, M.C., Corley, R.P., Krauter, K.S., Hewitt, J.K., (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics,* 96:684-695.

Zinbarg, R. E., & Barlow, D. H. (1996). Structure of anxiety and the anxiety disorders: A hierarchical model. *Journal of Abnormal Psychology, 105*(2), 181-193.

Table 1

Mapping of PTSD Symptoms onto Subfactors

| Symptom | | 4-DSM | 4-ALT |
|---|---|---|---|
| B1 | Intrusive thoughts | R | R |
| B2 | Recurrent dreams | R | R |
| B3 | Flashbacks | R | R |
| B4 | Emotional reactivity | R | R |
| B5 | Physical reactivity | R | R |
| C1 | Avoiding thoughts | A | A |
| C2 | Avoiding reminders | A | A |
| C3 | Amnesia for aspects | N | D |
| C4 | Loss of interest | N | D |
| C5 | Detachment | N | D |
| C6 | Restricted affect | N | D |
| C7 | Foreshortened future | N | D |
| D1 | Sleep disturbance | H | D |
| D2 | Irritability | H | D |
| D3 | Difficulty concentrating | H | D |
| D4 | Hypervigilance | H | H |
| D5 | Exaggerated startle | H | H |

Note: P = PTSD, R = reexperiencing, A = avoidance, N = numbing, H = hyperarousal, D = dysphoria. 4-DSM = four factor model based on Diagnostic and Statistical Manual-IV definition of PTSD; 4-ALT = four factor model equivalent to 4-DSM except 3 nonspecific hyperarousal items join numbing items to form a dysphoria factor.

Table 2

Ordinal Variable Frequencies

| % | 0 | 1 | 2 |
|---|---|---|---|
| SAD | 75.0 | 22.8 | 2.2 |
| GAD | 85.4 | 11.3 | 3.3 |
| MDD | 86.5 | 7.1 | 6.4 |
| PTSD | 91.5 | 7.8 | 0.7 |
| ADHD | 53.8 | 41.7 | 4.6 |
| ODD | 80.8 | 15.0 | 4.2 |
| CD | 51.1 | 38.0 | 10.9 |
| DRUG | 78.1 | 21.9 | - |
| DSM-Reexperiencing | 93.2 | 6.9 | - |
| DSM-Avoidance | 95.4 | 4.6 | - |
| DSM-Numbing | 94.8 | 5.2 | - |
| DSM-Hyperarousal | 96.2 | 3.8 | - |
| ALT-Dysphoria | 93.8 | 6.2 | - |
| ALT-Hyperarousal | 98.8 | 1.2 | - |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders; DSM = PTSD subfactors based on Diagnostic and Statistical Manual; ALT = Alternative PTSD subfactors. N = 3867.

Table 3

Phenotypic Correlations

| | SAD | GAD | MDD | PTSD | ADHD | ODD | CD | DRUG |
|---|---|---|---|---|---|---|---|---|
| SAD | 1 | | | | | | | |
| GAD | .41 | 1 | | | | | | |
| MDD | .31 | .44 | 1 | | | | | |
| PTSD | .32 | .37 | .31 | 1 | | | | |
| ADHD | .26 | .36 | .32 | .23 | 1 | | | |
| ODD | .26 | .37 | .44 | .26 | .48 | 1 | | |
| CD | .25 | .19 | .29 | .15 | .32 | .43 | 1 | |
| DRUG | .20 | .16 | .26 | .33 | .29 | .39 | .56 | 1 |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders; DSM = PTSD subfactors based on Diagnostic and Statistical Manual; ALT = Alternative PTSD subfactors. N = 3532.

Table 4

Phenotypic Correlations by Gender

| | SAD | GAD | MDD | PTSD | ADHD | ODD | CD | DRUG |
|---|---|---|---|---|---|---|---|---|
| SAD | 1 | .37 | .32 | .32 | .23 | .21 | .24 | .27 |
| GAD | .45 | 1 | .45 | .36 | .33 | .39 | .21 | .19 |
| MDD | .28 | .46 | 1 | .30 | .36 | .52 | .31 | .29 |
| PTSD | .34 | .43 | .32 | 1 | .21 | .24 | .17 | .33 |
| ADHD | .28 | .40 | .29 | .26 | 1 | .45 | .31 | .26 |
| ODD | .32 | .36 | .43 | .31 | .53 | 1 | .38 | .35 |
| CD | .27 | .19 | .28 | .17 | .36 | .50 | 1 | .58 |
| DRUG | .15 | .12 | .20 | .35 | .34 | .42 | .58 | 1 |

Note: Correlations within males are below the diagonal, correlations within females are above the diagonal. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders; N = 3867.

Table 5

Exploratory Factor Analysis Disorder Loadings

| | Two Factor Solution | | | | Three Factor Solution | | |
|---|---|---|---|---|---|---|---|
| | Including DRUG | | Excluding DRUG | | | | |
| | 1 | 2 | 1 | 2 | 1 | 2 | 3 |
| SAD | **.52** | .00 | **.55** | .01 | **.55** | .00 | .03 |
| GAD | **.86** | -.22 | **.74** | .00 | **.73** | -.02 | .02 |
| MDD | **.58** | .08 | **.42** | .28 | **.42** | .00 | **.30** |
| PTSD | **.47** | .05 | **.53** | -.01 | **.53** | .09 | .28 |
| ADHD | **.43** | .21 | .19 | **.47** | .19 | .00 | **.47** |
| ODD | **.41** | **.36** | .00 | **.83** | .01 | -.01 | **.82** |
| CD | .01 | **.75** | -.01 | **.53** | -.01 | .15 | **.50** |
| DRUG | -.01 | **.74** | - | - | .00 | **2.14** | .00 |
| Factor Correlations | | | | | | | |
| 1 – 2 | .52 | | .59 | | .13 | | |
| 1 – 3 | - | | - | | .58 | | |
| 2 - 3 | - | | - | | .23 | | |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. Path coefficients greater than .30 are displayed in bold font. N = 3532.

Table 6

Exploratory Factor Analysis Disorder Loading by Gender and Sample

| | Full Sample (n = 3867) | | | | One Individual per Family (n = 1729) | | | |
|---|---|---|---|---|---|---|---|---|
| | Male | | Female | | Male | | Female | |
| | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 |
| SAD | **.51** | .05 | **.44** | .07 | .14 | **.30** | **.47** | .02 |
| GAD | **.96** | -.23 | **.80** | -.19 | -.22 | **1.11** | **.70** | -.16 |
| MDD | **.50** | .13 | **.69** | .01 | .27 | **.34** | **.73** | -.07 |
| PTSD | **.45** | .13 | **.42** | .08 | .24 | .19 | **.44** | .18 |
| ADHD | **.32** | **.36** | **.46** | .13 | **.41** | .25 | **.47** | .02 |
| ODD | .27 | **.55** | **.54** | .17 | **.68** | .08 | **.68** | .06 |
| CD | -.11 | **.81** | .02 | **.72** | **.83** | -.17 | .22 | **.55** |
| DRUG | -.17 | **.82** | -.05 | **.83** | **.79** | -.17 | -.10 | **1.05** |
| Factor Correlations | | | | | | | | |
| 1 – 2 | .57 | | .58 | | .53 | | .51 | |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. For each disorder, the larger path coefficients are displayed in bold font.

Table 7

Confirmatory Factor Analysis Model Fits

| | Chi-sq | df | p | CFI | TLI | RMSEA | RMSR |
|---|---|---|---|---|---|---|---|
| 1 Factor | 1512.04 | 20 | 0 | .79 | .71 | .15 | .08 |
| 2 Factor INT-EXT | 984.86 | 19 | 0 | .86 | .80 | .12 | .06 |
| INT-EXT, ADHD and ODD on INT | 865.22 | 19 | 0 | .88 | .82 | .11 | .06 |
| INT-EXT, ADHD and ODD on Both | 603.56 | 17 | 0 | .92 | .86 | .10 | .05 |
| 3 Factor, CD on 2nd and 3rd Factors | 476.17 | 16 | 0 | .94 | .89 | .09 | .04 |

Note: df = degrees of freedom; p = probability; CFI = comparative fit index; TLI = Tucker-Lewis Index; RMSEA = root mean square error of approximation; RMSR = root mean square residual. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. N = 3532.

Table 8

Confirmatory Factor Analysis Measurement Invariance Across Gender

| | Gender | $\chi^2$ | df | p | AIC | RMSEA | Δp |
|---|---|---|---|---|---|---|---|
| 1 Factor | equal | 1073.42 | 64 | 0 | 945.42 | .15 | <.01 |
| 1 Factor | free | 1019.71 | 56 | 0 | 907.71 | .17 | |
| 2 Factor INT - EXT | equal | 879.17 | 63 | 0 | 753.17 | .12 | <.01 |
| 2 Factor INT - EXT | free | 815.63 | 54 | 0 | 707.63 | .11 | |
| 2 Factor ADHD and ODD on INT | equal | 726.88 | 63 | 0 | 600.88 | .10 | <.01 |
| 2 Factor ADHD and ODD on INT | free | 657.76 | 54 | 0 | 549.76 | .10 | |
| 2 Factor ADHD and ODD on Both | equal | 640.42 | 61 | 0 | 518.42 | .10 | <.01 |
| 2 Factor ADHD and ODD on Both | free | 535.47 | 50 | 0 | 435.47 | .09 | |
| 3 Factor CD on 2nd and 3rd | equal | 594.69 | 60 | 0 | 474.69 | .09 | <.01 |
| 3 Factor CD on 2nd and 3rd | free | 506.01 | 48 | 0 | 410.01 | .09 | |

Note: $\chi^2$ = chi-square; df = degrees of freedom; p = probability; AIC = Aikake's Information Criterion; RMSEA = root mean square error of approximation;. Δp = p value of chi-square difference test comparing models with each gender's parameter estimated constrained to be equal versus free to differ. INT = internalizing; EXT = externalizing; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder. N = 1729.

Table 9

Correlations Between PTSD Subfactors and Disorders

| | SAD | GAD | MDD | ADHD | ODD | CD | DRUG | Average of INT Disorders | Average of EXT Disorders |
|---|---|---|---|---|---|---|---|---|---|
| Reexperiencing | .27 | .40 | .36 | .27 | .42 | .24 | .37 | .34 | .33 |
| Avoidance | .29 | .42 | .33 | .26 | .43 | .27 | .38 | .35 | .33 |
| DSM - Numbing | .30 | .40 | .28 | .24 | .33 | .21 | .31 | .33 | .27 |
| DSM - Hyperarousal | .30 | .43 | .34 | .27 | .42 | .21 | .34 | .35 | .31 |
| Alt - Dysphoria | .29 | .39 | .27 | .25 | .37 | .21 | .32 | .32 | .29 |
| Alt - Hyperarousal | .33 | .37 | .33 | .18 | .30 | .15 | .36 | .34 | .25 |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. INT = 3 internalizing disorders of SAD, GAD, and MDD; EXT = 4 externalizing disorders of ADHD, ODD, CD, and DRUG. DSM = Diagnostic and Statistical Manual; ALT = alternative model. n = 1729.

Table 10

PTSD Subfactor Models

| | INT | | EXT | | Both | | |
|---|---|---|---|---|---|---|---|
| | $\chi^2$ | Estimate | $\chi^2$ | Estimate | $\chi^2$ | INT Estimate | EXT Estimate |
| PTSD* | **984.86** | .53 | 1234.41 | .44 | 984.82 | .54 | -.01 |
| Reexperiencing | 443.18 | .61 | 505.57 | .55 | **424.08** | .42 | .21 |
| Avoidance | 444.90 | .62 | 501.43 | .56 | **420.75** | .41 | .24 |
| DSM - Numbing | **418.31** | .56 | 519.87 | .47 | 415.02 | .48 | .09 |
| DSM - Hyperarousal | 449.93 | .62 | 549.23 | .54 | **440.34** | .48 | .15 |
| ALT - Dysphoria | 439.78 | .56 | 508.57 | .49 | **428.72** | .42 | .16 |
| ALT - Hyperarousal | **517.77** | .56 | 662.12 | .45 | 517.68 | .57 | -.02 |

Note: INT = internalizing; EXT = externalizing; $\chi^2$ = chi-square; DSM = Diagnostic and Statistical Manual; ALT = alternative. Model with best combination of fit and parsimony is highlighted in bold font. * n for PTSD analyses = 3532, n for all subfactor analyses = 1729.

Table 11

Within-Trait, Cross-Sib Correlations

| | MZ | DZ | Sib |
|---|---|---|---|
| SAD | .42 | .18 | .23 |
| GAD | .37 | .09 | .16 |
| MDD | .39 | .04 | .25 |
| ADHD | .37 | .16 | .12 |
| ODD | .43 | .14 | .06 |
| CD | .57 | .33 | .23 |
| PTSD | .27 | .28 | .18 |
| DRUG | .83 | .57 | .54 |

Note: MZ = monozygotic; DZ = Dizygotic; Sib = full sibling. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. N = 3532.

Table 12

Cross-Trait, Cross Sibling Correlations

| | MZ | DZ | Sib |
|---|---|---|---|
| SAD – GAD | .24 | .08 | .10 |
| SAD - MDD | .24 | .00 | .05 |
| SAD – ADHD | .18 | .11 | .00 |
| SAD - ODD | .25 | .08 | .01 |
| SAD - CD | .16 | .06 | .04 |
| SAD - PTSD | .29 | .16 | .12 |
| SAD - DRUG | .15 | .13 | .17 |
| GAD - MDD | .32 | .05 | .06 |
| GAD - ADHD | .22 | .07 | .04 |
| GAD - ODD | .22 | .14 | .11 |
| GAD - CD | .06 | .03 | .06 |
| GAD - PTSD | .31 | .10 | .08 |
| GAD - DRUG | .12 | .05 | .08 |
| MDD - ADHD | .21 | .11 | .11 |
| MDD - ODD | .22 | .13 | .07 |
| MDD - CD | .22 | .05 | .06 |
| MDD - PTSD | .25 | .17 | .08 |
| MDD - DRUG | .14 | .18 | .10 |
| ADHD - ODD | .27 | .17 | .11 |
| ADHD - CD | .27 | .12 | .10 |
| ADHD - PTSD | .25 | .04 | .04 |
| ADHD - DRUG | .20 | .17 | .12 |
| ODD - CD | .31 | .16 | .05 |
| ODD - PTSD | .24 | .19 | .12 |
| ODD - DRUG | .29 | .27 | .18 |
| CD - PTSD | .27 | .09 | .12 |
| CD - DRUG | .45 | .34 | .23 |
| PTSD - DRUG | .25 | .21 | .13 |

Note: MZ = monozygotic; DZ = Dizygotic; Sib = full sibling. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorders. N = 3532.

Table 13
Genetic Model Fits

| | $\chi^2$ | df | p | AIC | RMSEA |
|---|---|---|---|---|---|
| ACE Cholesky | 503.78 | 44 | 0 | 415.78 | 0 |
| ACTE Cholesky | 469.07 | 8 | 0 | 453.07 | 0 |
| ADE Cholesky | 560.15 | 44 | 0 | 472.15 | 0 |
| 2 Common A | 604.54 | 63 | 0 | 478.54 | 0 |
| 1 Common C | 542.04 | 64 | 0 | 414.04 | 0 |
| 2 Common E | 892.44 | 63 | 0 | 766.44 | 0 |
| Post Hoc Model 1 | 593.01 | 62 | 0 | 469.01 | 0 |
| Post Hoc Model 2 | 544.61 | 65 | 0 | 414.61 | 0 |

Note: $\chi^2$ = chi square; df = degrees of freedom; p = probability; AIC = Aikake's Information Criterion; RMSEA = root mean square error of approximation; A = additive genetic influences; C = shared environment; D = dominant genetic influences; T = twin environment; E = nonshared environment. Post hoc model 1 contains 3 common genetic factors: the first loads on internalizing disorders, the second on Attention Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, and Conduct Disorder; the third on Conduct Disorder and Drug Use Disorder Symptoms. Post hoc model 2 contains three common genetic factors: the first loads on all disorders except Drug Use Disorder symptoms, the second loads on Posttraumatic Stress Disorder and externalizing disorders, the third loads on Conduct Disorder and Drug Use Disorder Symptoms.

Table 14

Variance Decomposition

| | A | C | E |
|---|---|---|---|
| SAD | .20 | .17 | .62 |
| GAD | .20 | .10 | .71 |
| MDD | .24 | .11 | .65 |
| PTSD | .20 | .12 | .67 |
| ADHD | .26 | .07 | .67 |
| ODD | .20 | .10 | .69 |
| CD | .48 | .07 | .45 |
| DRUG | .54 | .29 | .17 |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorder Symptoms. A = additive genetic influences; C = shared environment; E = nonshared environment.

Table 15

Covariance Decomposition

| | Phenotypic Correlation | % of Covariation Due To… A | C | E |
|---|---|---|---|---|
| *Internalizing* | | | | |
| SAD - GAD | .41 | .49 | .10 | .41 |
| SAD - MDD | .31 | .71 | -.06 | .35 |
| SAD - PTSD | .32 | .55 | .26 | .19 |
| GAD - MDD | .44 | .49 | .05 | .47 |
| GAD - PTSD | .37 | .46 | .15 | .39 |
| MDD - PTSD | .31 | .61 | .15 | .24 |
| *Externalizing* | | | | |
| ADHD – ODD | .48 | .47 | .11 | .43 |
| ADHD - CD | .32 | .81 | .03 | .17 |
| ADHD - DRUG | .29 | .50 | .21 | .30 |
| ODD - CD | .43 | .57 | .07 | .36 |
| ODD - DRUG | .39 | .38 | .35 | .26 |
| CD - DRUG | .56 | .59 | .20 | .21 |
| *Internalizing - Externalizing* | | | | |
| SAD - ADHD | .26 | .64 | .00 | .36 |
| SAD - ODD | .26 | .50 | .17 | .33 |
| SAD - CD | .25 | .42 | .16 | .42 |
| SAD - DRUG | .20 | .00 | .73 | .27 |
| GAD – ADHD | .36 | .45 | .04 | .50 |
| GAD - ODD | .37 | .35 | .22 | .43 |
| GAD - CD | .19 | .53 | .02 | .46 |
| GAD - DRUG | .16 | .00 | .52 | .48 |
| MDD - ADHD | .32 | .57 | .14 | .29 |
| MDD - ODD | .44 | .32 | .16 | .52 |
| MDD - CD | .29 | .39 | .19 | .42 |
| MDD - DRUG | .26 | .00 | .48 | .52 |
| PTSD - ADHD | .23 | .95 | -.09 | .14 |
| PTSD - ODD | .26 | .74 | .24 | .03 |
| PTSD – CD | .15 | 1.34 | .26 | -.60 |
| PTSD - DRUG | .33 | .30 | .40 | .30 |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorder Symptoms. A = additive genetic influences; C = shared environment; E = nonshared environment.

Table 16

Path Coefficients

| | $A_1$ | $A_2$ | $A_3$ | $A_4$ | $A_5$ | $A_6$ | $A_7$ | $A_8$ |
|---|---|---|---|---|---|---|---|---|
| SAD | .45 | | | | | | | |
| GAD | .44 | - | | | | | | |
| MDD | .49 | - | - | | | | | |
| PTSD | .39 | - | - | .22 | | | | |
| ADHD | .37 | - | - | .34 | - | | | |
| ODD | .29 | - | - | .35 | - | - | | |
| CD | .23 | - | - | .52 | - | - | .40 | |
| DRUG | - | - | - | .42 | - | - | .27 | .53 |
| | $C_1$ | $C_2$ | $C_3$ | $C_4$ | $C_5$ | $C_6$ | $C_7$ | $C_8$ |
| SAD | .42 | | | | | | | |
| GAD | .10 | .30 | | | | | | |
| MDD | -.04 | .08 | .32 | | | | | |
| PTSD | .20 | .13 | .14 | .00 | | | | |
| ADHD | .00 | .05 | .13 | -.20 | .23 | | | |
| ODD | .10 | .24 | .17 | -.09 | .08 | -.01 | | |
| CD | .09 | -.02 | .18 | .00 | -.06 | .15 | .00 | |
| DRUG | .34 | .14 | .39 | .00 | .02 | .09 | .00 | .00 |
| | $E_1$ | $E_2$ | $E_3$ | $E_4$ | $E_5$ | $E_6$ | $E_7$ | $E_8$ |
| SAD | .79 | | | | | | | |
| GAD | .21 | .81 | | | | | | |
| MDD | .14 | .22 | .76 | | | | | |
| PTSD | .08 | .16 | .04 | .78 | | | | |
| ADHD | .12 | .19 | .05 | -.01 | .79 | | | |
| ODD | .11 | .17 | .24 | -.05 | .19 | .75 | | |
| CD | .13 | .07 | .11 | -.18 | .02 | .13 | .63 | |
| DRUG | .06 | .07 | .14 | .13 | .07 | .05 | .13 | .31 |

Note: SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention Deficit / Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; DRUG = Drug Use Disorder Symptoms. A = additive genetic influences; C = shared environment; E = nonshared environment.

Figure 1

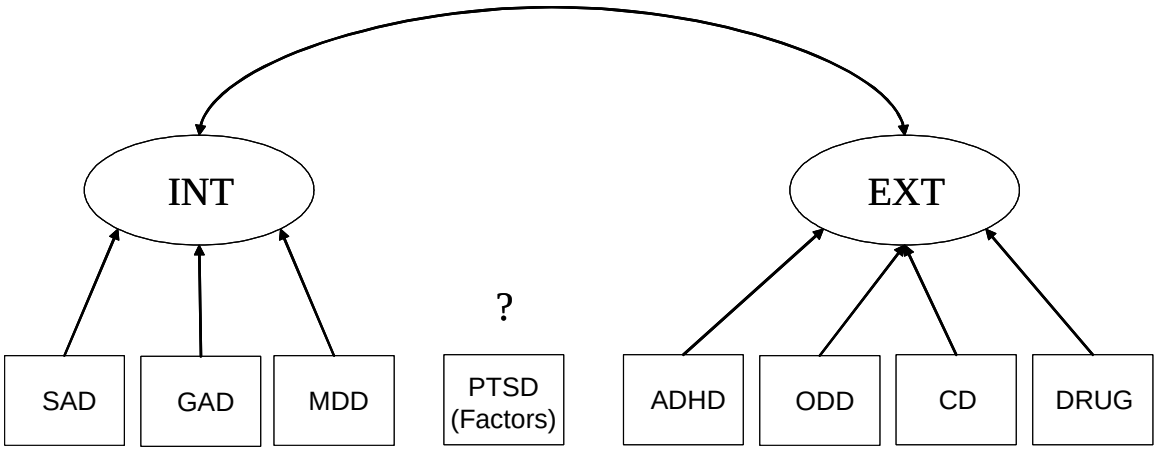

Figure 2

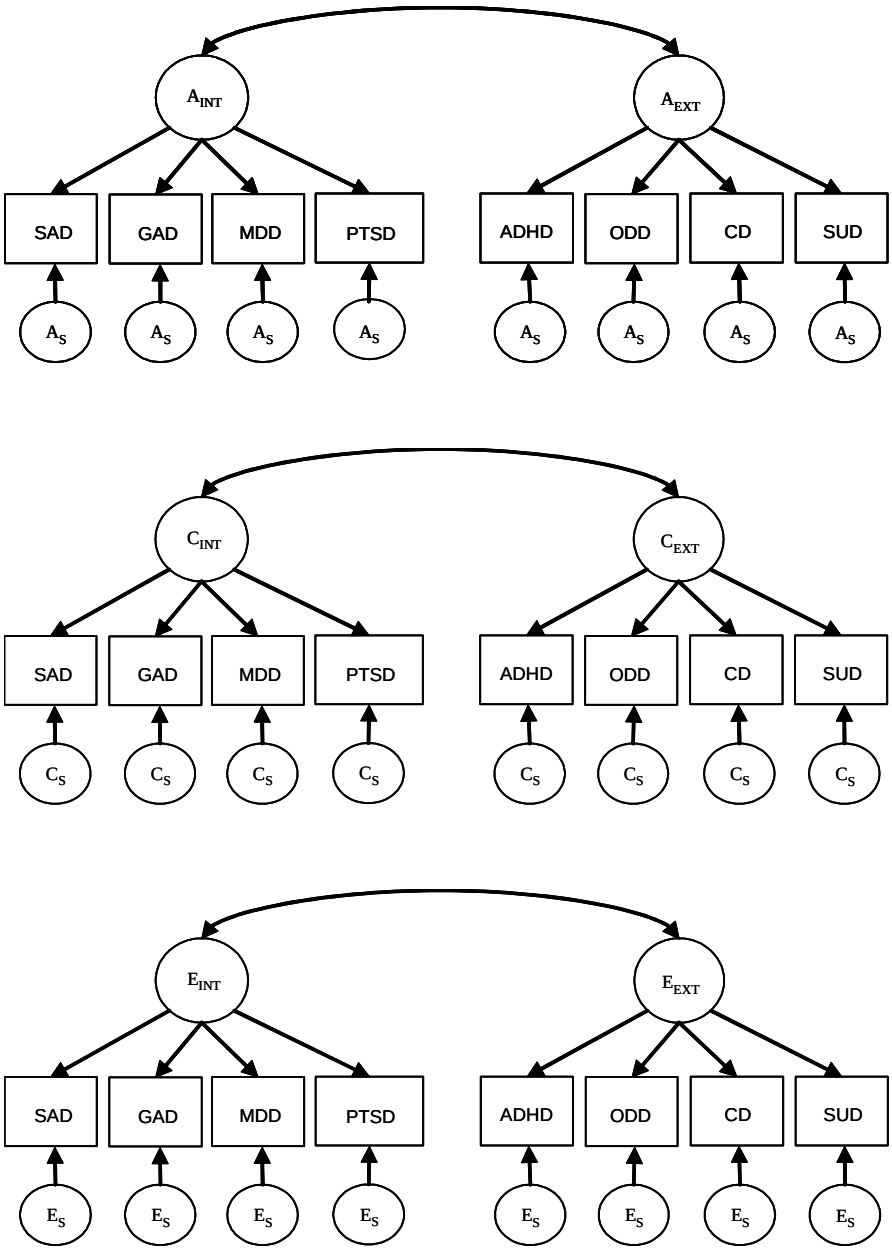
A_INT
A_EXT
SAD
GAD
MDD
PTSD
ADHD
ODD
CD
SUD
A_S
C_INT
C_EXT
C_S
E_INT
E_EXT
E_S

Figure 3

Best Fitting Model

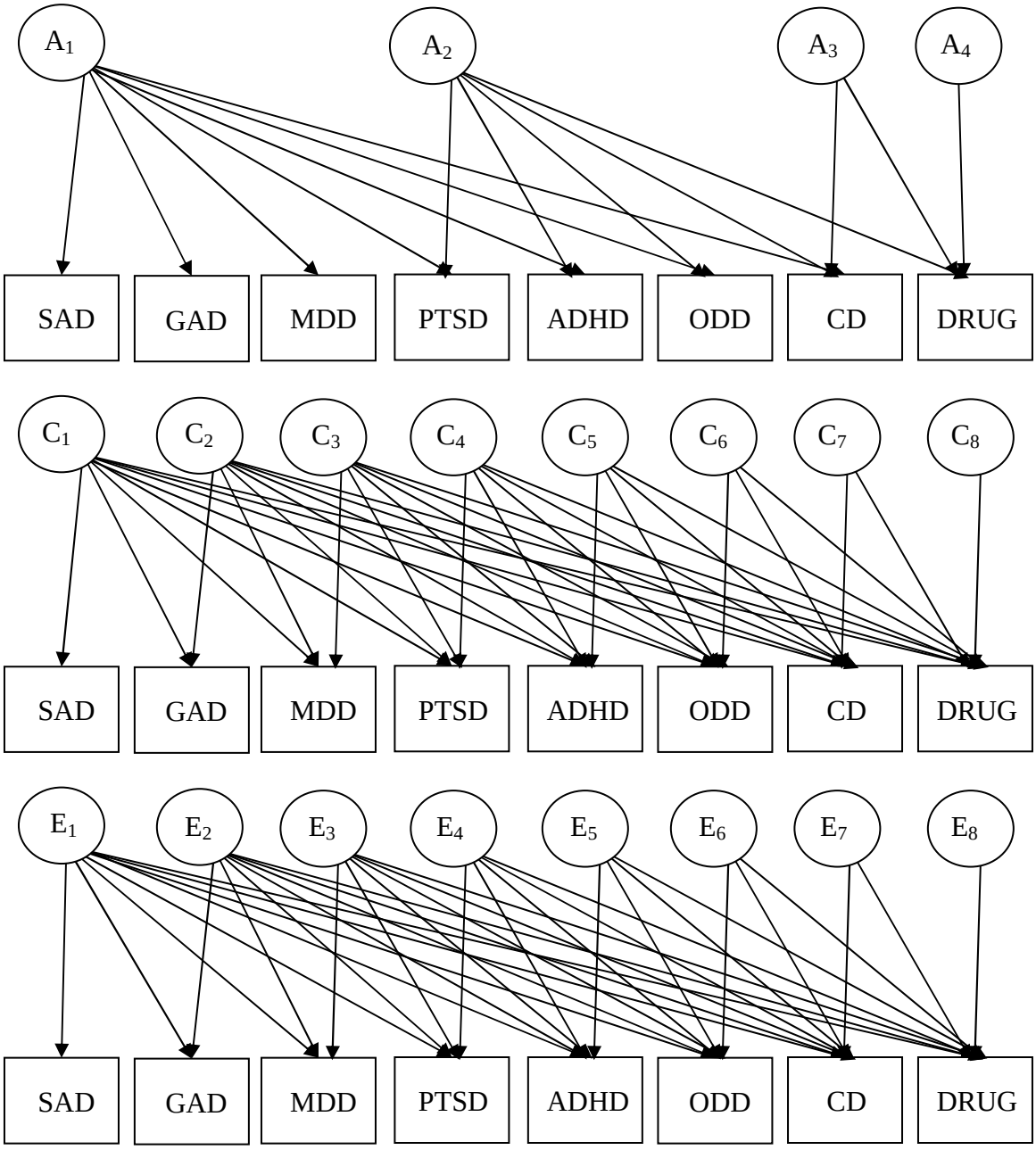

Figure Captions

Figure 1

Models assessing the fit of PTSD and its subfactors. Models in which PTSD and its subfactors load on INT, on EXT and on both are compared. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; CD = Conduct Disorder; ODD = Oppositional Defiant Disorder; ADHD = Attention-Deficit Hyperactivity Disorder; SUD = Substance Use Disorder; INT = Internalizing; EXT = Externalizing.

Figure 2

Multivariate twin model with two common genetic, two common shared environmental, and two common nonshared environmental factors, and disorder-specific paths. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; ADHD = Attention-Deficit Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; SUD = Substance Use Disorder; INT = Internalizing; EXT = Externalizing. A = additive genetic influences; C = shared environmental influences; T = twin environmental influences; E = nonshared environmental influences. Subscript S = disorder-specific;

.

Figure 3

Multivariate twin model with three common genetic factors (A) and disorder-specific genetic influences on DRUG. Shared environmental influences (C) and nonshared environmental influences (E) are Cholesky decompositions. SAD = Separation Anxiety Disorder; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Posttraumatic Stress Disorder; CD = Conduct Disorder; ODD = Oppositional Defiant Disorder; ADHD = Attention-Deficit Hyperactivity Disorder; DRUG = drug use disorder symptoms.